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A

PRACTICAL TREATISE

ON

MEDICAL DIAGNOSIS

FOR STUDENTS AND PHYSICIANS.

BY

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all probability not tuberculous. They are not found in mucopurulent but in putrid sputum.

The smegma bacilli stain with most of the reagents used in demonstrating tubercle bacilli, but are unable to resist decolorization by a 30 per cent. solution of nitric acid in water. The tubercle bacilli are, however, considered more resistant to decolorizing agents. After staining with carbol-fuchsin, if the specimen is treated with saturated alcoholic solution of methylene-blue for three to five minutes, the smegma bacilli will usually give up their stain. Or if the specimen stained with carbol-fuchsin is treated with 5 per cent. HCl for three minutes, then 70 per cent. alcohol for ten minutes, and finally with Gabbet's methylene-blue, the tubercle bacilli withstand the decolorizing and remain stained. Smegma bacilli are smaller, are not grouped like tubercle bacilli, and do not show beading.

**PNEUMOCOCCUS—DIPLOCOCCUS PNEUMONIÆ; MICROCOCCUS LAN-
CEOLATUS.** The causative factor in most cases of acute croupous pneumonia in its typical form is a paired lancet-shaped coccus, often irregular in size, with a tendency to chain formation. Frequently oval or conical forms are present, and there is apt to be variation in the size of the cocci forming the pair. The organism has a distinct capsule. In the sputum of croupous pneumonia these pneumococci are usually present in large numbers. Their presence within leucocytes and their tendency to chain formation have been especially noted in such cases.

Staining the Pneumococcus. Pneumococci are stained in cover-glass preparations with the ordinary aniline dyes, as given above. The capsule may be stained and differentiated in the same way, but it more often requires a special method. Welch recommends the following: Dried cover-glass preparations are treated first with glacial acetic acid, which is allowed to drain off, and then (without washing in water) with aniline oil gentian-violet solution. (See under Tubercle Bacilli.) The staining solution is repeatedly added to the surface of the cover-glass until all of the acid is displaced. The specimen is now washed in a weak salt solution (about 2 per cent.), and examined in the same, not in balsam. The capsule and coccus can then be differentiated. Sputum stained by Gram's method, thoroughly decolorized by alcohol, and counterstained with a watery solution of eosin, or a 1 per cent. aqueous solution of aurantia, has been found satisfactory for photomicrographic work. Degenerative and involution-forms of the pneumococcus are constantly met with. There will be variations in size and shape, and the capsule may contain only the remains of a coccus, or it may be entirely empty.

Significance of the Pneumococcus. This micro-organism is found in nearly all cases of acute croupous pneumonia, and in many cases of bronchopneumonia. Its presence has also been observed in health in the saliva. It is found also in acute pleuritis, endocarditis, pericarditis, peritonitis, acute purulent meningitis, and otitis media. Its presence in empyema is considered of favorable import. It has also been found in cases of synovitis, osteomyelitis, and abscess formation in various situations. It may cause a general septicæmia—*i. e.*, pneumococcus septicæmia.

BACILLUS CAPSULATUS MUCOSUS OR FRIEDLANDER'S BACILLUS CAPSULATUS. This organism is found in the sputum in health in a certain number of cases. In association with the pneumococcus it can cause pneumonia. It can also produce pneumonia by itself in rare instances.

In three fatal cases of pneumonia due to the capsule bacillus alone there have been found in the sputa large numbers of capsule bacilli. These were frequently inside of leucocytes, and many alveolar cells were filled with these bacilli.

BACILLUS OF INFLUENZA. This organism is found in the sputum in cases of influenza or influenza pneumonia. It was first isolated from the sputum by Pfeiffer. The organism appears as a small bacillus with rounded ends. Its length varies somewhat, and thread-like involution-forms may appear. It *stains* more deeply at the ends than at the middle, and the long forms may show irregularity of staining.

WHOOPING-COUGH BACILLI. Minute bacilli have been discovered in the sputum in cases of whooping-cough by Czplewski, Koplik, Zusch, and others. At present the results are not sufficiently uniform to prove that these bacilli are of etiological import in the disease.

ACTINOMYCES. When the lung or pleura is infected by this fungus, actinomyces may be found in the sputum. The disease in these organs is rare. Macroscopically they appear as small kernels, yellowish white or greenish yellow, and having the shape of a millet-seed. Under the microscope they are recognized by the rounded, club-like bodies projecting from all sides of an unformed central mass. They are seen better when not stained. (See page 810.)

CHEMISTRY OF SPUTUM.

As the chemical examination of the sputum does not aid us in diagnosis, it has little or no value. *Mucin*, *nuclein*, and *serum-albumin* are constituents of sputum in health. *Peptone* is present whenever there is pus, and is especially marked in pneumonia. *Volatile fatty acids*, such as butyric and acetic, occur at times, markedly so in pulmonary gangrene. *Glycogen* has been obtained by Solomon, and a *ferment* resembling one of the pancreatic ferments has been detected, especially in pulmonary gangrene and putrid bronchitis. Of inorganic substances, *chlorides of sodium and magnesium*; *phosphates of sodium, lime, and magnesium*; *sulphates of sodium and lime*; *carbonate of sodium, lime, and magnesium*; and in a few cases *phosphate of iron* and *silicates* have been obtained. (Von Jaksch.)

CHAPTER XLIII.

THE URINE.

Inspection of Urine.

THE urine is not simply an index of the condition of the kidneys. It varies, within the bounds of health, in color, quantity, and quality. Food, exercise, and other conditions modify the secretion. It can readily be seen, therefore, that any general disease and many local diseases cause alterations in the character of the urine. Any abnormality of the urine may be symptomatic of renal disease or of disease beyond the point at which the urine passes out of the body. Usually abnormal changes in the urine, due to the general condition, do not give rise to local renal symptoms or to abnormal renal function. The exception is seen when an excess of uric acid, of urates, or of oxalates is passed. They may give rise to local pain and may set up sufficient irritation to cause albuminuria.

The urine in health is a clear yellow or amber-colored fluid, having a specific gravity of about 1020, and generally acid in reaction. It contains normally about 45 parts in the 1000 of solid matter, the principal part of which is urea— $21\frac{1}{2}$ parts. The other solids are uric acid and its salts; certain extractives—creatin, creatinin, ammonia, hippuric acid, xanthin, hypoxanthin, sarcin, pigment, etc.; and chlorides, phosphates, sulphates, with their bases, soda, potash, lime, and magnesia.

The *volume* of urine passed in twenty-four hours is usually from 40 to 50 ounces, but it may fall to 30 ounces or rise to 70 without the existence of disease. Women are believed to pass from 5 to 10 ounces less than men. The volume is diminished when the skin is acting freely, as in warm weather, and when the bowels are loose; and, on the other hand, cold, constipation, and nervous excitement, especially if it induce anxiety and fear, all tend to increase the quantity secreted.

Color. The *color* of the urine is due largely but not wholly to urobilin, which is formed from the hæmatin of the blood. The color deepens when the urine is concentrated, which occurs after a hearty meal, or exercise, especially in warm weather; and it becomes paler when a large quantity is passed. The color is frequently changed in disease. In fevers the urine soon after being passed is apt to become *turbid* from the precipitation of urates, and the color varies from white, especially in children, to yellow, brown, or pink. When the precipitate settles, the supernatant urine may be high-colored and clear, or slightly opaque from some suspended matter.

The admixture of pus and chyle gives the urine a *milky* color. The urine may also be *yellowish white* and turbid from phosphates, semen. sarcinæ, and bacteria.

The urine is *red*, reddish brown, or “smoky” in acute nephritis, the color being due to blood. It is bloody in hæmaturia, cancer of the kidneys and bladder, and in injuries of the genito-urinary apparatus. The urine is very red and clear when concentrated and containing a large amount of urates. The red color of the urine may be due to hæmoglobin, constituting *hæmoglobinuria*, or to excess of urobilin, as in scurvy and pernicious anæmia. Hæmoglobinuria occurs as the result of the action of certain poisons, such as potassium chlorate; in infectious diseases, such as scarlet fever; and in malarial fevers; also in a peculiar disease known as paroxysmal hæmoglobinuria.

Again, a *golden-red* discoloration of the urine is common in jaundice; frequently the upper layers have a greenish tinge by reflected light.

Finally, a *red color* is produced by the internal administration of log-wood and fuchsin.

A *yellow* color, when opaque, may be due to suspended phosphates and urates. Urine is sometimes of a golden-yellow or saffron color in jaundice, and from the effects of santonin, picric acid, and rhubarb taken internally. A yellow or yellowish-white turbidity may be due also to a mixture of pus and phosphates, and sometimes to semen, sarcinæ, and bacteria. The urine usually becomes more or less opaque and yellow when it has undergone alkaline fermentation. Such a change occurs normally within a longer or shorter time after the urine has been passed. It is promoted by heat and exposure to air, and retarded by cold and exclusion from air. If possible, the urine should be examined before this fermentation has occurred. Pathologically, in cases of cystitis, the urine when passed is already in a state of alkaline fermentation.

The urine is sometimes *chocolate-brown* when it contains blood and the blood has been acted upon by the urine, producing methæmoglobin.

Brown, greenish-brown, or black urine may result from contained bile-salts; from indican; from carbolic acid, creosote, and tar used internally or externally; from the internal use of senna, and in cases where there are melanotic tumors. Senator injected melanin into human beings and obtained in four cases only a large indicanuria.

Urine is *pale* usually in proportion as it is copious in quantity. It is paler in those who are using milk or vegetable diet than in those who eat meats. Under the influence of nervous excitement, especially anxiety and the dread of an approaching ordeal, such as an examination, an abnormal quantity of very pale urine is secreted.

Pathologically, *pale* urine is characteristic of diabetes, chronic Bright's disease, and polyuria. Such urine is also secreted in hysterical attacks, at the crises of febrile diseases, and in anæmic conditions.

Quantity. The volume may be increased, diminished, or unchanged in disease. It is *increased* principally in three diseases—diabetes mellitus, diabetes insipidus, and in the middle period of chronic Bright's disease, especially in the interstitial form. In diabetes mellitus it sometimes exceeds 32 pints. It may be increased also in hypertrophy of the left ventricle, which induces greater pressure in the renal arteries as well as in the whole arterial system; and also in cystic degeneration, and in double hydronephrosis.

The urine is *diminished* in acute nephritis and in the final stages of chronic nephritis; sometimes also it is diminished in the middle period of chronic nephritis, but usually it is here increased. All diseases which directly or indirectly impair the force of the circulation lessen the secretion of the urine. Hence, the quantity is diminished in diseases of the heart muscle and in valvular diseases not fully compensated; in emphysema and in chronic bronchitis. It is lessened also in cirrhosis of the liver. In febrile diseases the urine is scanty and high-colored, and sometimes it is almost suppressed (anuria).

The urine is sometimes *suppressed* in acute nephritis, such as follows scarlet fever, and in the final stages of all the organic affections of the kidneys—chronic nephritis, hydronephrosis and pyonephrosis, etc. It may result (1) from the destruction of the secreting tissue of the kidney or interference with its nervous or vascular supply, or (2) from mechanical obstruction to the outflow of urine. To the first class belong the cases of suppression occurring in acute and chronic nephritis, and those due to shock and collapse, whether occurring in the collapse stage of yellow fever, cholera, and other grave febrile diseases, or from serious internal injuries.

Such suppression sometimes follows slight operations on the urethra (urethral fever); or results from the internal administration of drugs, the excretion of which occasions violent irritation of the kidney—such as cantharides, turpentine, and even the inhalation of ether. Clinically, suppression not due to obstruction is distinguished from the obstructive form by the character of the urine, which is usually not entirely suppressed, and by the more rapid course of the disease. The urine, according to Roberts, is either concentrated or it contains albumin, blood, and casts. Death or recovery results within a day or two. In the obstructive form, on the other hand, the urine which escapes past the obstacle is pale, watery, and devoid of albumin and casts.

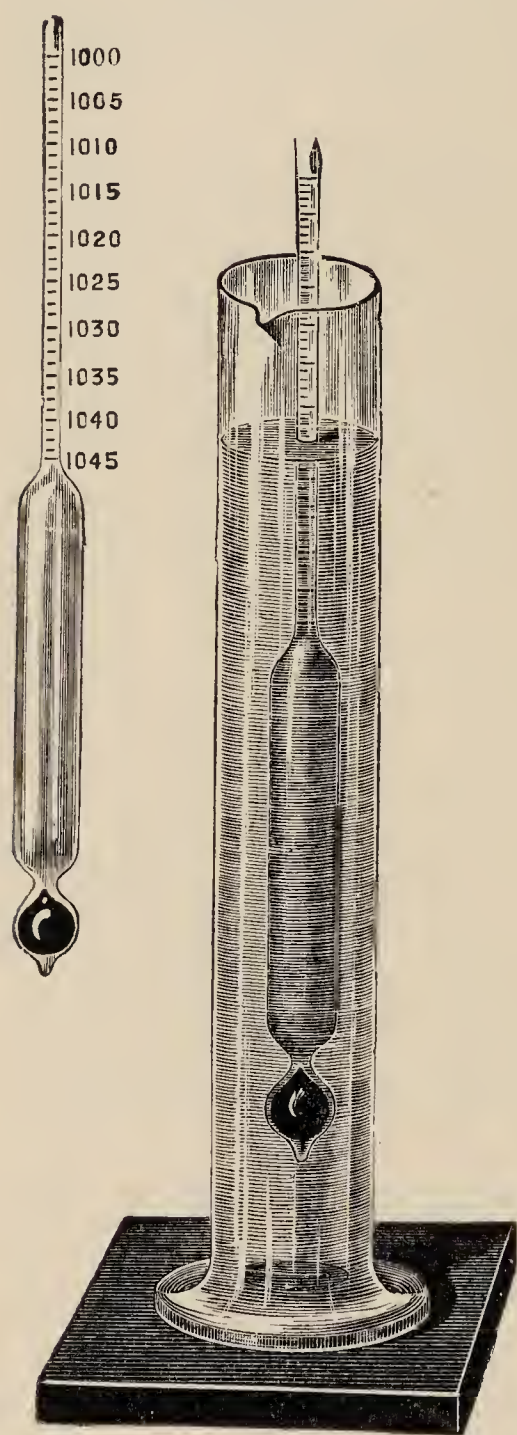
Obstructive suppression is the result of the plugging of the ureter by a calculus when the opposite kidney is either absent or incapable of secreting. It also results from the occlusion of the ureters by morbid growths especially at the vesical orifices, from lateral pressure upon the ureters, or from some interference with or malformation of the ureters or renal arteries.

Acute transient obstructive suppression occurs sometimes in persons with enlarged prostates or old strictures who have drunk too freely of alcoholic beverages, and perhaps have wound up a debauch by sexual intercourse.

Density. The average density of normal urine is about 1020. It may fall to 1015 or rise to 1025, depending upon the quantity of fluid and food taken, the condition of the atmosphere, especially as regards temperature, and upon mental influences usually of an emotional character. The specific gravity of the urine is tested by a urinometer graduated for degrees of density between 1000 and 1040. Only a reliable instrument should be used. As the density of the urine passed at different times during the day varies greatly, the urine for the whole twenty-four hours should be saved and a specimen of this tested.

The method of taking the specific gravity is very simple. A test-tube or graduate, having a diameter of about $1\frac{1}{4}$ inches and a length of 6 or 7 inches, is filled with urine to such a point that the lowest part of the urinometer, when inserted, floats clear of the bottom of the tube. The instrument must also float free of the sides of the tube. The specific

FIG. 220.



Urinometer. (W. SIMON.)

gravity should then be read off from below—that is to say, by holding the tube up so that the level of the fluid is a little above that of the eye. Most *urinometers* are graduated for 60° F., but in ordinary examinations it is not necessary to have the urine exactly at this temperature; it should, however, be allowed to cool after it has been passed, otherwise the specific gravity will appear to be too low.

In *disease* the specific gravity varies more widely than in health; it may fall to 1005 or 1000 in diabetes insipidus and chronic Bright's disease, and rise to 1060 or even higher in diabetes mellitus. As a rule, to which the urine in diabetes mellitus is the principal exception, the color is an index of the density; pale urine being of low density and high-colored urine of high density.

The density is increased when the urine is scanty in amount, whether as the result of fever, acute nephritis, large consumption of solid food, exercise, or free sweating. In all such cases the specific gravity rarely rises above 1035, and usually not above 1028 or 1030. When the specific gravity rises above 1035, and the urine is pale in color, the presence of sugar is to be suspected; and when it rises above 1040, sugar is almost certainly present.

The specific gravity is lowered by drinking copiously, by the effect of external cold, by a diet of vegetables and milk, and in general by the same causes that make the urine copious.

Usually, but not always, a urine containing a large amount of albumin is of low density.

Pathologically, a low specific gravity is encountered in diabetes insipidus, in which it may fall nearly or quite to 1000; generally in the middle or quiescent period of chronic Bright's disease; in the crisis of fevers; in obstructive suppression; in hysterical attacks, and in hydro-nephrosis.

Specific Gravity as an Index of the Amount of Solids. If the last two figures of the specific gravity be doubled, the sum will represent the amount of solid matter in 1000 grains of urine. This is Trapp's method; the estimate is only approximate, but it is useful. Of course, the urine for twenty-four hours must be used.

Reaction. The reaction of *healthy urine* is usually *acid*, but it may be neutral or slightly alkaline about two hours after a meal of mixed food. The acidity is tested with litmus-paper: the blue paper is turned purple or red by an acid, and the red paper is turned blue by an alkali. Violet paper is to be preferred, as it is suitable for showing both reactions, an alkali turning it blue and an acid red.

The acidity of the urine is *increased* in gout, lithiasis, acute rheumatism, diabetes, chronic Bright's disease, and as the result of the administration of vegetable or mineral acids.

The urine is *alkaline* because of alkaline fermentation in the bladder in cystitis; from the presence of much blood or pus; from prolonged immersion of the body in a cold bath; in debilitating diseases and in some cases of nervous dyspepsia, and as the result of the internal administration of alkalies.

Urinary Sediments. A white flocculent sediment, composed of epithelium and mucus, occurs normally in most urines after they have stood for some hours.

A dense sediment, varying in color from that of brown sugar to pink or red, consists of amorphous urates. It dissolves upon the application of heat. A sediment usually resembling red pepper, but sometimes of a brown color, consists of uric acid, the presence of which may be proved by the murexide test. The suspected material is placed in a crucible or evaporating-dish with a few drops of nitric acid. As heat is applied the uric acid or amorphous urate dissolves with effervescence. Heat is now kept up until the material is evaporated to dryness; it is then allowed to cool. If it is now touched with a glass rod that has been dipped in strong ammonia, a characteristic blue or violet color is produced. Uric acid is not usually so abundant as the sediment of amorphous urates; it sinks more rapidly, and is deposited from acid, high-colored urines.

A yellowish or whitish sediment may consist of sodium urate.

A white sediment usually consists of phosphates, associated with which we sometimes find a white sediment consisting of ammonium urate, with or without pus. Such urines are alkaline. A white sediment may be due to uric acid, especially in children.

A yellowish-white sediment may consist of pus, with or without mucus. If the urine is acid, the sediment is loose and free to move; but when the urine is alkaline, the sediment consists of a viscid, coherent mass, which can be drawn out into tough, stringy filaments.

A chocolate-brown sediment, occurring in a reddish, smoky urine, consists of blood from the kidneys. Clots of blood come from the ureters, bladder, or urethra.

Odor. The odor of normal urine is sometimes spoken of as aromatic, but generally it is sufficiently characteristic to be described best as urinous. When the urine is concentrated, the odor is intensified, and may become unpleasantly strong, like the urine of the horse.

Certain articles of food, such as garlic and asparagus, give the urine characteristic odors. Turpentine, both when taken internally and inhaled, gives to it the odor of violets. The odors of copaiba and cubebs can be detected in the urine of patients who are taking these drugs.

In marked cystitis the natural urinous odor becomes more pungent, and is blended with a strong ammoniacal odor. When much pus is present, and the urine has stood a while, a putrid odor is developed.

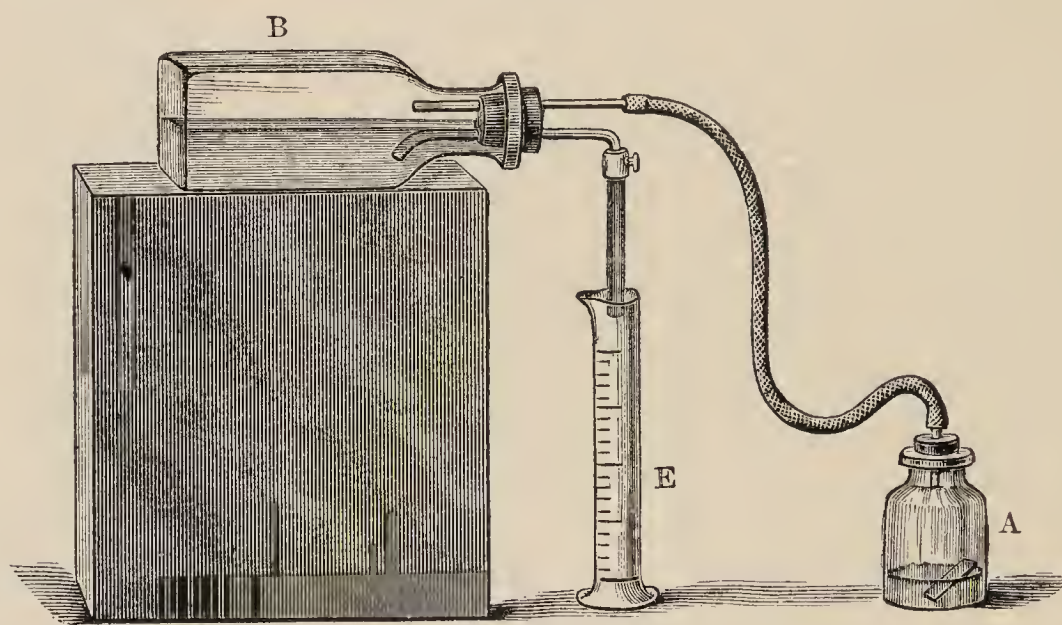
In diabetes mellitus the urine has a sweetish, hay-like odor. In diabetic coma the odor is sometimes like that of chloroform, due to the presence of acetone and diacetic acid in the urine. This odor, however, is more likely to be detected in the breath.

Chemical Examination of the Urine.

Examination of the urine by the unaided senses, which has been dwelt upon thus far, is simply preliminary to an examination by chemical methods and by instruments of precision, particularly the microscope.

Urea. Urea is freely soluble in water, and hence never appears as a sediment. It is the most important final product of nitrogenous disintegration in the body, and is an index of the eliminative power of the kidneys. Usually the density of the urine increases in proportion to the amount of urea contained in it. The average daily amount of urea excreted by an adult man between the ages of twenty and forty years is

FIG. 221.



Squibb's ureometer.

about 500 grains. The urea, like the total volume of the urine, is subject to variations within the limits of health. It is increased after a meal, especially if the latter be rich in nitrogenous food; after copious ingestion of liquids, and by a close atmosphere. On the other hand, fasting, free perspiration, a loose condition of the bowels, and a vegetable or milk diet diminish the quantity of urea. Again, the quantity varies with the age of the person. According to Ralfe, at five years the daily amount is 180 grains; at twelve, 320; at twenty-one, 535, and at forty years, 555 grains.

A large man will excrete absolutely more than a small man, and a large muscular man will excrete relatively more than a fat man of the same height.

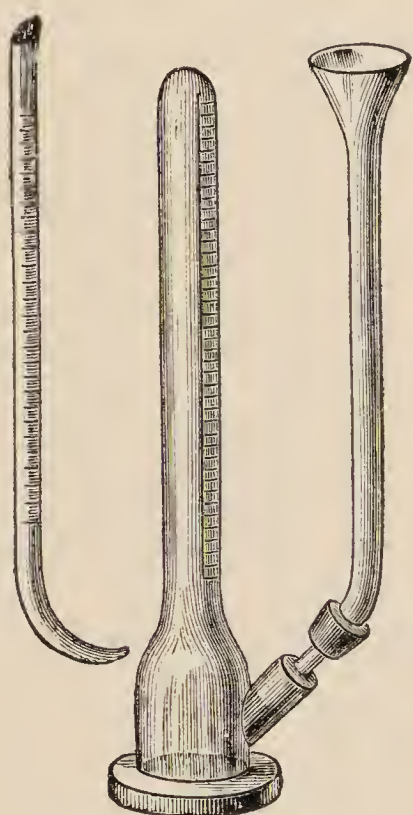
The excretion of urea is increased in fever and inflammatory diseases;

in diabetes mellitus and insipidus; in malaria, pernicious anæmia, and after a crisis in pneumonia. It is increased also by certain beverages, as coffee, and by many drugs, especially those which act as hepatic stimulants.

It is diminished in all forms of nephritis, especially when uræmia results; in acute gout and chronic rheumatism; in disease accompanied by emaciation and cachexia; in leprosy, pemphigus, melancholia, imbecility, catalepsy, hysteria, and cholera. (Saundby.)

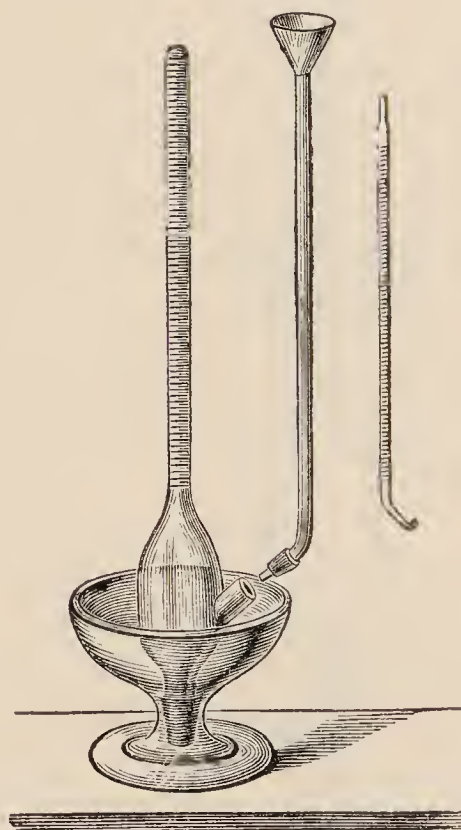
Estimation of Urea. For the methods employed in the exact quantitative estimation of urea the student is referred to special works on the urine.

FIG. 222.



Green's ureometer.

FIG. 223.



Marshall's ureometer.

For ordinary clinical purposes the apparatus devised by Professor Charles Doremus, and known by his name, gives sufficiently accurate results. The principle upon which it is based is that urea when brought in contact with sodium hypobromite is decomposed, and free nitrogen is eliminated. The nitrogen evolved is the measure of the urea contained in the urine. The instruments are graduated so that each division of the scale represents 1 grain of urea per fluidounce of urine.

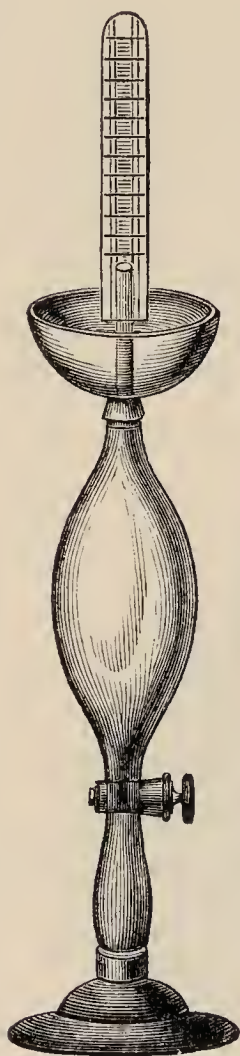
Specimens should be taken from the mixed amount of twenty-four hours on account of well-known variation at different times.

The hypobromite solution is prepared by dissolving 100 grammes of sodium hydroxide in 250 c.c. of water, cooling the solution, and then adding 25 c.c. of bromine.

It is better, however, to prepare the hypobromite solution freshly for each examination. This can readily be done by having a solution of sodium hydroxide containing 6 ounces to a pint of water. It should be kept tightly corked with a rubber or paraffined stopper. The sodium hydroxide solution is poured into the long tube of the ureometer to the mark =, then $\frac{1}{10}$ its volume of bromine is introduced by means of a

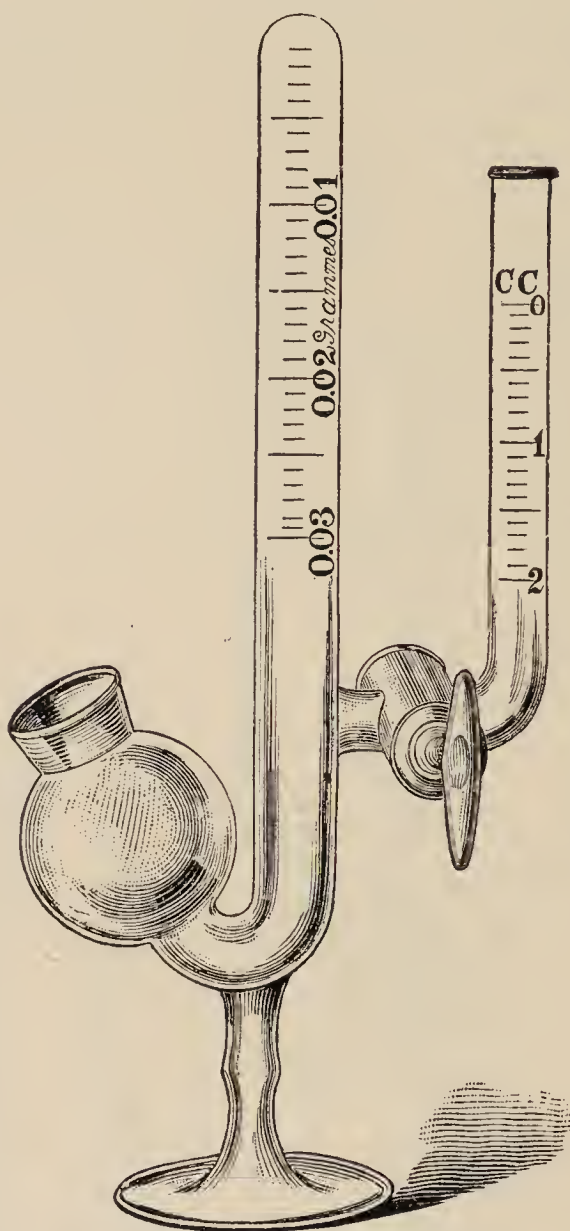
pipette, and sufficient water added to fill the long arm and the bend of the tube. The hypobromite solution should fill the tube completely, and any bubbles rising to the top of the tube should be removed before the introduction of the urine. The pipette is then filled with the urine up to the 1 c.c. mark, any urine adhering to its surface being carefully wiped off. The pipette is introduced carefully, so as not to compress the bulb

FIG. 224.



Hüffner's ureometer.

FIG. 225.



Doremus' ureometer.

until the point extends as high up as possible beyond the bend. The bulb is now compressed slowly until 1 c.c. of urine has been introduced. Decomposition of the urea occurs and bubbles of nitrogen rise to the surface of the long arm of the tube; when bubbles of gas cease to be evolved, the volume of nitrogen gas is read off, and according to the graduations on the tube considered as so many grains of urea per fluid-ounce of urine, or as so many milligrammes of urea in 1 c.c. of urine, according to whether it is graduated in the English or in the metric system.

Chlorides. The presence or absence of chlorides is sometimes of diagnostic value. They are increased when absorption of exudates or transudates is going on, and in malarial fevers, diabetes insipidus, and Bright's disease. They are diminished or absent in pneumonia during its progressive stage, and in fevers. The chlorine of the chlorides can be detected and roughly estimated by an 8 or 10 per cent. solution

of argentic nitrate. A few drops of nitric acid are first added to the urine, to prevent the silver from precipitating phosphoric acid. A single drop of the silver solution mentioned will precipitate the chloride of the chlorides in a thick white lump, which falls to the bottom of the test-tube, provided the amount present is normal. If, on the other hand, the quantity is diminished to 0.1 per cent. or less, it will not be precipitated in a lump or lumps, but a white cloudiness is produced which renders the whole solution opaque. If no precipitation or cloudiness occurs, the chlorides are absent.

Sulphates. The daily excretion of sulphuric acid is about $2\frac{1}{2}$ grammes, chiefly in the form of sodium and potassium salts—inorganic sulphates. The organic sulphates are combinations with phenol, cresol, indol, skatol, etc.

Test for Inorganic Sulphates. To 10 c.c. of urine acidulated with a few drops of hydrochloric acid, 3 c.c. of a 10 per cent. barium chloride solution are added. If the normal amount of sulphates is present, an opaque milkiness develops; if the precipitate is thick and creamy, the sulphates are in excess; if the specimen merely becomes opalescent, they are diminished.

An increase in the percentage of sulphates when not due to the ingestion of phenol and allied substances for medicinal purposes, indicates the presence of a putrefactive process in the body, as when there is a putrid abscess or retention of the intestinal contents.

Qualitative Tests for Albumin. *Serum-albumin* is of common occurrence, but can not ever be looked upon as a normal constituent of the urine, though its presence by no means indicates disease of the kidney. While it is the ordinary form of albumin present in the urine, other proteids, as globulin, mucin, peptone, albumose, fibrin, and also hæmoglobin and methæmoglobin, are found at times. The most trustworthy tests for ordinary albumin (*serum-albumin*) are: boiling with the addition of nitric or acetic acid; overlaying cold nitric acid with urine (Heller's test); the picric acid, the potassium ferrocyanide, and the potassium mercuric iodide (Tanret's) tests. The author believes that many of the recent tests, such as sodium tungstate, acidulated brine, magnesium nitrate, phenic-acetic acid, and trichloroacetic acid, are too sensitive and precipitate other substances in the urine, and therefore are not reliable for clinical work. *Serum-globulin* responds to all the following tests for *serum-albumin*. Its differentiation is not difficult, but usually unnecessary. (See note on page 650.)

Boiling and Nitric Acid Test. A narrow, long test-tube is filled two-thirds full of urine and the upper third boiled thoroughly, and then a few drops of nitric acid are added. Any albumin present will be coagulated and appear as a white cloud, contrasting strongly with the clear unboiled urine beneath it. When the albumin is moderate or even small in amount, it can be detected without difficulty by simply holding the test-tube up to the light. When there is only a faint trace present, it will be overlooked unless the tube be examined against a dark surface in such a way that the light falls upon it from above, in front, and preferably a little to one side. A cloud may escape detection when

looked for by artificial light, but may be distinct by daylight. Serum-globulin is also precipitated by this test. But serum-globulin is not often present by itself, and its significance is not yet understood. It may be detected in any urine, as Roberts points out, by diluting the urine with pure water, the urine then becoming more or less milky. It may be removed from urine by saturating the latter with magnesium sulphate and filtering off the precipitated globulin. The presence of serum-globulin in no way interferes with the test for serum-albumin.

If the urine is opaque from amorphous urates, it is unnecessary to filter them out; heat much below boiling will dissolve them, the precipitation of albumin occurring later at a higher temperature.

If the urine is alkaline or faintly acid, phosphates will produce a cloud upon heating the urine; but they are instantly dissolved upon the addition of a few drops of nitric or acetic acid.

Mucin produces an opalescence upon heating with an organic acid, but Saundby declares that it coagulates not in flocculi, as is the case with albumin, but in the form of tiny filaments.

Boiling and Acetic Acid Test. This is preferred by many to the preceding test. It is performed in a similar manner. Acetic acid is, however, not reliable for acidulation; it precipitates the mucin which is often found in healthy urine, forming a white cloud which is apt to be mistaken for albumin; this is especially true in urines of high specific gravity containing uric acid, urates, or oxalates.

The Nitric Acid (Heller's) Test. This test, while not so delicate as the acetic acid test, is very simple and accurate in its results. Cold nitric acid is poured into a test-tube to the depth of about an inch. The tube is then inclined to an angle of about 45 degrees, and urine allowed to flow gently down upon the acid by trickling along the side of the tube from a pipette or glass tube. At the point of contact of the acid and urine a zone of white, coagulated albumin forms. The test can also be made as follows: Into a short, broad test-tube several cubic centimetres of urine are poured; nitric acid is introduced with a pipette provided with a rubber bulb by passing the pipette through the urine to the bottom of the tube and gently pressing the rubber bulb; care must be taken to withdraw the pipette as soon as the last portion of acid is expelled, so that no air-bubbles will break up the point of contact of the urine and acid. The thickness of the white zone is generally an index of the amount of albumin present. If there is barely a trace of albumin, half an hour may be required to develop any opalescence.

A cloud of urates is sometimes produced and obscures the test. This cloud does not, however, begin at the point of contact and extend upward, but at the upper level of the urine and extends downward, and is dissipated by heat.

Patients who are taking copabia or cubebs pass a urine which gives a white zone at the point of contact with cold nitric acid, but heat diminishes the opacity, and the precipitate is soluble in alcohol; the odor of the drugs in the urine assists in the detection of their presence.

The Picric Acid Test. This is an extremely delicate test for albumin. A saturated solution of picric acid is allowed to flow down upon and

slightly mix with the upper layers of the urine, which half fills a good-sized test-tube. At the point of contact an opaque white zone of coagulated albumin is formed. If no white zone appears, albumin is almost certainly absent. Hence, the picric acid test is a valuable negative test. But, unfortunately, a white zone is formed by peptone, mucin, and various alkaloids, particularly quinine. The white zone produced by the presence of the substances just named disappears upon the application of heat, whereas an opalescence due to albumin becomes diffused throughout the whole urine.

The Potassium Ferrocyanide Test. This test is highly recommended as simple, rapid, and accurate by Purdy, who performs it as follows: Into a test-tube are poured 15 to 30 drops of acetic acid, and then two or three times that amount of potassium ferrocyanide solution (1 to 20) is added, and the two thoroughly mixed by shaking the tube. The urine is now added to the depth of two-thirds of the test-tube. If any albumin is present, it will be precipitated throughout the whole volume of urine in the form of a milky-white flocculent cloud, the intensity depending on the amount of albumin present. By this method all modifications of albumin, acid or alkaline, are precipitated and the precipitation of mucin is avoided. It gives no reaction with phosphates, urates, peptones, vegetable alkaloids, or the acids found in the urine after the ingestion of copaiba, etc. This test may also be performed as follows: An ordinary test-tube is half filled with urine and a drachm or two of the potassium ferrocyanide solution (1 to 20) are added. After thoroughly mingling the reagent and the urine, a few drops of acetic acid are added. If albumin is present, it will plainly come into view. This test, therefore, depends upon the production of a cloudiness or milkiness throughout the entire mixture in the tube. To some eyes the albumin is not so readily perceived as in those tests which depend upon the formation of a distinct line at the point of contact.

The Potassio-mercuric Iodide (Tanret's) Test. The solution is made as follows: potassium iodide, 3.32 grammes; mercuric chloride, 1.35 grammes; acetic acid, 20 c.c.; distilled water, about 30 c.c. (the potassium iodide and the mercuric chloride should be dissolved separately in the water and the solution mixed, to which the acetic acid is added and the whole made up to 60 c.c. with distilled water). As thus prepared, the test is applied by the contact-method by overlaying the reagent with urine. This test responds to all modifications of albumin, also to peptones and proteoses, as well as to the vegetable alkaloids and acids found in the urine after the ingestion of copaiba, etc. All reactions except those occurring with albumin, mucin, and the acids found in the urine after the ingestion of copaiba, etc., disappear with heat. It is a very good and delicate control-test for albumin. The solution, however, is of a yellowish hue, quite similar to the color of urines of low specific gravity; this sometimes renders the line of contact difficult to perceive.

It is well to follow a routine method in testing for albumin: first, by boiling and the addition of nitric acid, and then the contact-test (Heller's); if there is a doubt, either the potassium ferrocyanide or picric acid test; finally, Tanret's solution will reveal minute quantities of albumin, and may be used as a confirmatory test.

In all the tests for albumin mentioned a clear urine is necessary, especially when the amount of albumin is very small. This can be obtained by filtration when the opacity is due to pus, blood, mucus, or uric acid; and more effectively by the addition of a small quantity of sodium hydroxide, warming slightly, and filtering. If the filtrate is not clear, a few drops of magnesium fluid (magnesium sulphate, pure ammonium chloride, and pure liquor ammoniæ, of each, 2 drachms; distilled water, 2 ounces), as recommended by Hoffmann and Ultzmann, may be added, and the urine again warmed and filtered.

RÉSUMÉ OF TESTS FOR ALBUMIN.

I. The *heat* test.

A. Method: Albumin is precipitated on boiling.

B. Exceptions: 1. In alkaline urines albumin may be overlooked from the formation of soluble potassium and magnesium compounds. When patients are taking alkaline salts, the test may be fallacious.

2. An excess of acid may also interfere with the test.

3. Feebly alkaline or neutral urines produce a precipitate of earthy phosphates, but it is instantly soluble in a small quantity of acid.

4. Patients on a vegetable diet pass urine containing carbonates which precipitate with heat. The addition of an acid causes great evolution of gas.

II. The *heat and acetic acid* test.

Method: Determine the reaction of the urine. If alkaline, make faintly acid with acetic acid; then boil and add a little more acetic acid. If there is no precipitate, boil again. The acetic acid precipitates nucleoproteids, which are excluded by the methods above described.

III. The *heat and nitric acid* test.

A. Method: Bring the urine to the boiling-point and add nitric acid drop by drop, shaking the mixture between each addition. A small precipitate is thrown down even if a very small amount of albumin is present. The nitric acid should not exceed more than one-tenth of the volume of urine examined. The urine must not be heated after the addition of the acid.

B. Exceptions: 1. In concentrated urines, uric acid or its salts may precipitate. Distinguish from albumin by filtering off the precipitate and testing it by the biuret reaction, or dilute the urine with an equal volume of water when uric acid will not precipitate.

2. Resin acids in turpentine, benzoin, cubebs, and other balsams, if present in the urine, are precipitated by nitric acid. Distinguish from albumin by adding 1 or 2 volumes of alcohol when the solution is cool. The precipitate of resin acids is dissolved.

3. In urines containing biliverdin a precipitate is formed. Distinguish from albumin by adding alcohol, which dissolves biliverdin.

IV. *Cold nitric acid* test.

A. Method: Pour the urine gently on the nitric acid. The albumin coagulates in the presence of an excess of strong nitric acid. A ring appears at the surface of contact if albumin is present. A second ring may be seen $\frac{1}{10}$ to 1 cm. above the junction, due to nucleoproteids. Distinguish from albumin by repeating the test with urine diluted with 2

or 3 volumes of water. The albumin ring diminishes and the nucleoproteid ring is unchanged or increased. A haze due to nucleoproteid may form, and also continue after dilution.

B. Exceptions: 1. In concentrated urines a secondary ring due to uric acid may form above the junction. It is soluble on gently heating, and does not form when the urine has been diluted.

2. In highly concentrated urine a precipitate of urea nitrate may fall. Distinguish by its crystalline nature.

3. Resin acids cause a precipitate of uniform cloudiness. Distinguish by solubility in alcohol.

4. In highly colored urines the urinary pigments form a colored ring at the plane of contact, and in bilious urines the play of colors, as in Gmelin-Malin-Heintz's test for bile, is seen.

5. The urine of patients taking alkaline iodides gives a dense brown ring of iodine. Distinguish by adding a few cubic centimetres of chloroform and mixing them. A violet tinge is imparted to the liquid.

6. Albumoses are precipitated, as well as all forms of albumin. Distinguish by the previously mentioned tests. Peptone and vegetable alkaloids are not precipitated.

V. The *potassium ferrocyanide* and *acetic acid* test.

A. Method: It is best performed as a ring test. The urine should be carefully run into a mixture of 20 or 30 drops of acetic acid and 60 or 90 drops of saturated solution of potassium ferrocyanide. A white ring forms at the junction if albumin is present. With small amounts of albumin the ring takes some minutes to form.

B. Exceptions: 1. Albumoses are precipitated. They are soluble in excess of acetic acid. They disappear on heating and reappear on cooling.

2. Resin acids give a precipitate which is soluble in alcohol.

3. Phosphates, urates, alkaloids, and peptones are not precipitated.

VI. Roberts' *brine* test.

Saturated sodium hydrate solution with 5 per cent. hydrochloric acid. It does not darken the urine nor precipitate uric acid.

A. Method: Use the ring test, which shows albumin and albumoses.

B. Exceptions: Resin acids precipitate. Distinguish by dissolving in alcohol.

VII. The *salt* and *acetic acid* test.

The acetic acid is substituted for HCl, and a large excess of salt solution used.

A. Method: The salt solution is first added to the urine and thoroughly mixed. Acetic acid is then poured in. Nucleoproteids are not precipitated. (All other forms of albumin are precipitated.) Salt and vinegar may be used, and the mixture heated in a metal spoon.

B. Exceptions: 1. Albumoses form and disappear on heating, to reappear on cooling.

2. If albumoses and albumin appear together, boil for a short time and filter the hot fluid through a warm filter. The clear filtrate becomes turbid from albumoses as it cools.

3. Resin acids and uric acid are precipitated, the latter only in concentrated urines and after standing. Distinguish by the usual tests.

V. and VI. do not generally precipitate nucleoproteids. With VII., if equal parts of urine and salt solution are used with a few drops of acetic acid, nucleoproteids are not precipitated. The solution must be boiled when test VII. is employed.

VIII. *Salicylsulphonic acid* test.

All forms of albumin are precipitated. The precipitate becomes flocculent on heating. If the urine is alkaline more of the reagent is needed than if acid. Phosphates, urates, bile, alkaloids, and drugs do not give a reaction.

A. Method: After adding the solution to the urine, heat and allow to stand.

B. Exceptions: Albumoses are precipitated, but disappear on heating and reappear on cooling.

IX. *Trichloroacetic acid* test.

Exceptions: 1. Precipitates uric acid when in excess. Distinguish by heating, which dissolves the acid, or by diluting the urine before applying the test.

2. Nucleoproteids give an opalescence. Albumoses are not precipitated.

X. *Picric acid* test.

A. Method: A saturated solution of picric acid must be used alone, or in combination with hydrochloric or acetic acid. Value doubtful.

B. Exceptions: Uric acid, creatinin, nucleoproteids, alkaloids, potassium salts, and albumoses are precipitated.

XI. *Millard's reagent*.

Value doubtful. Precipitates albumoses, nucleoproteids, alkaloids, and resin acids. Distinguish by usual tests.

XII. *Tanret's reagent*.

Very delicate. Precipitates all forms of albumin, albumoses, nucleoproteids, peptones, alkaloids, and resin acids. Distinguish by usual tests.

XIII. *Spiegler's reagent*.

Delicate. Precipitates albumin, albumoses, and nucleoproteids, but not peptones.

XIV. *Acetic acid* test.

Method: Filter the urine and add acetic acid to a portion, pouring the two in the tube held against a black background. Albumin and nucleoproteids are precipitated. Distinguish by diluting the filtered urine with 2 or 3 volumes of distilled water, then add acetic acid, and compare the precipitate with that in an undiluted specimen. A nucleoprotein precipitate will increase in intensity. An albumin precipitate will diminish or remain unchanged.

Salicylsulphonic acid is the most delicate test. An objection to it is the fact that it precipitates nucleoproteids. Control the test by Heller's cold nitric acid test, from which the nucleoproteids are removed as above described.

Quantitative Estimation of Albumin. The most direct method is by coagulating the albumin by boiling, collecting it upon a weighed filter, washing with water and finally with alcohol, drying and weighing it. Such a process, however, consumes too much time for clinical purposes,

and it is not faultless. An approximate estimation may be made by boiling the urine in a test-tube, adding several drops of nitric acid, allowing the albumin to settle, and then comparing the depth of albumin with the height of the column of urine. In this way we may speak of urine furnishing one-tenth or one-quarter of its bulk of coagulated albumin.

Esbach has invented an *albuminometer* (Fig. 226) which gives good results. The solution used to precipitate the albumin consists of 10 grammes of picric acid and 20 grammes of citric acid, chemically pure and dry, dissolved in 900 c.c. of hot water; and after cooling, diluting the solution to 1000 c.c. The urine is diluted with a definite amount of water if it contains too much albumin. The albuminometer is filled to the mark **U** with urine, and from that mark to **R** with the reagent. The tube is then corked with a rubber stopper, turned upside down 10 times, so as to mix the urine intimately with the reagent, and then allowed to stand undisturbed for twenty-four hours. At the end of that time the depth of the sediment of coagulated albumin is ascertained by observing where the top of the sediment comes in contact with a mark on the scale on the tube. Each mark corresponds to 0.1 per cent. of albumin.

FIG. 226.

Esbach
albuminometer.

This estimation, as already stated, is not absolutely accurate. Nevertheless, if used systematically, and always in the same way, relative values will be obtained, and these are the most important in watching the progress of a case, as they give positive information regarding an increase or diminution of the amount of albumin in the urine. It scarcely need be said that the urine tested must be a portion of the whole twenty-four hours' urine.

The estimation of the amount of albumin is also readily made with the centrifugal machine: to 10 c.c. of the albuminous urine are added 3.5 c.c. of potassium ferrocyanide solution (1 to 10) and 1.5 c.c. of acetic acid; the mixture is then revolved in the machine about three minutes, and the amount of precipitate read off.

Albuminuria. Albuminuria is not indicative of disease of any one organ, nor does it point to any general pathological condition. It occurs:

1. In diseases of the kidney: acute and chronic Bright's disease, amyloid disease, tuberculosis, cancer, abscess, and calculus.
2. In disturbances of the circulation: diseases of the heart and chronic pulmonary diseases, as emphysema; obstruction of the renal arteries or veins, cirrhosis of the liver, peritonitis, pregnancy, abdominal tumors; in passive congestion due to great weakness; in anæmia and Graves' disease.
3. In febrile and inflammatory diseases: in the eruptive and infectious fevers, in rheumatism, diphtheria, pneumonia, and gout.
4. In blood diseases: purpura, leucocythæmia, and scurvy.
5. From the poisonous action of drugs: lead, turpentine, and others.
6. In nervous disorders: concussion of the brain and cerebral hemorrhage, epilepsy, tetanus, and delirium tremens; as Pye-Smith remarks, it is doubtful whether albuminuria is caused by the nervous diseases.

7. In local extrarenal affections : pyelitis, cystitis, gonorrhœa, and leucorrhœa.

8. As a functional disturbance : in young persons, particularly of the male sex, after exercise, a special diet, or a cold bath. Albumin may be found after rising in the morning, or early after dinner, or toward evening. On account of its occurring only at certain times it has been called "cyclical" or "intermittent," and because there is no evident disease present, it is occasionally spoken of as "physiological" albuminuria.

Goodhart examined the urine of 1500 individuals and noted albumin in 272, or in 20 per cent. In 39 cases the albuminuria could not positively be said to be due to disease of the kidney. Of these 39, 26 were males and 13 females. In 32 of the 39 cases it was temporary, and in most of them it disappeared within forty-eight hours or sooner. In 2 cases there were oxalates in the urine; in 1 hæmoglobinuria; in 8 leucorrhœal discharges and discharges from other parts of the genital passages (see division 7); and in 17 a markedly neurotic temperament. These last he thinks the most typical cases of intermittent albuminuria; on the whole, he regards the condition as less common than has been supposed.

One variety of functional albuminuria is apparently due to irritation of the kidney produced by the excretion of oxalates and uric acid. The urine is of increased density, 1028, 1030, or higher, and contains uric acid or oxalate of lime, or both, and cylindroids. Tube-casts are uncommon. The albuminuria usually disappears under proper diet. This condition is sometimes called "*morbis Da Costæ*."

It is conceded that there may be albuminuria of renal origin without renal disease, but the diagnosis must be by exclusion, and can be reached safely only after extended observation. The most important elements in the diagnosis are : the age of the patient, unimpaired general health, a specific gravity of the urine normal or above normal; the fact that the albuminuria is influenced by diet and exercise, and that it tends to disappear under suitable regimen. The prognosis is favorable.

Mucin. Nucleo-albumin, or nucleoproteid, is nucleic acid and chondrosulphonic acid combined with a proteid. Sometimes, pathologically, taurocholic acid enters into the combination. This is not true mucus, but urinary mucus. It is present in the urine in health, being especially abundant in women from the admixture of the vaginal secretion, and in excess in inflammatory conditions of the urinary tract. It is distinguished from albumin by the fact that it gives a precipitate upon the addition of a vegetable acid, as acetic or citric. The precipitate is increased by removing the salts of the urine by dialysis, or by dilution of the urine with two or three volumes of distilled water, diminishing thereby the relative proportion of salts to mucus. It is precipitated by dilute mineral acids, but is soluble in concentrated mineral acids or dilute alkalies.

According to Roberts, the best method for the detection of mucin is by means of a saturated solution of citric acid, employed in the same manner as the contact-method of applying the nitric acid test for albumin. A small quantity of the urine is first put in a test-tube, and citric acid allowed to trickle down the sides of the tube until it forms a distinct layer below the column of urine. If mucin is present, there will gradu-

ally appear an opalescent zone immediately above the layer of acid. Acetic acid, mixed with one-third of its volume of glycerin, answers admirably as a test for mucin. Sometimes, when mucin is very abundant, the addition of an excess of acetic acid produces a marked milkiness in the urine, which is not discharged by boiling.

Blood. Urine containing blood is usually red in color or reddish brown and opaque, but it may be chocolate-brown if the blood is present in large quantity and has been acted upon by the urine. Such urine necessarily contains albumin.

Blood occurs in the urine from (1) *diseases of the kidney and urinary passages*, among which are Bright's disease, acute congestion of the kidney, renal calculus, cancer, tuberculosis; from ureteritis, cystitis, urethritis, and injuries; (2) from *general diseases*, such as the eruptive and intermittent fevers, scurvy, purpura, peliosis rheumatica, leucocythæmia, cholera; (3) from *adjacent organs*, as in menstruation and hemorrhage from the uterus; (4) from the *toxic action of drugs*—cantharides, turpentine, and other violent irritants of the kidney; (5) *vicariously*—occasionally menstruation fails to occur and hæmaturia replaces it. The same is true of bleeding from piles. Latour has reported a case of asthma which subsided suddenly upon the appearance of hæmaturia.

The chemical tests for blood are the same as those for its coloring-matter, and will be referred to under Hæmoglobin.

Hæmoglobin. Hæmoglobin is, of course, present whenever blood is, but sometimes it occurs independently of hæmaturia. Thus it is found in grave infectious diseases, as the result of the toxic action of drugs, such as carbolic acid, and in an independent disease known as paroxysmal hæmoglobinuria. A suitable test consists in adding 1 or 2 drops of freshly prepared tincture of guaiac to about 1 drachm of urine, then shaking the mixture and adding several drops of a solution of hydrogen peroxide. If blood-coloring matter be present, a beautiful blue coloration will be produced.

The same test answers for methæmoglobin and hæmatin.

Paroxysmal Hæmoglobinuria. The urine contains blood, or only the coloring-matter of the blood. Hæmoglobinuria is more frequent in adults; it may be excited by a cold bath, or exposure to cold, or by exertion. It is sometimes associated with Raynaud's disease. The attacks come on suddenly, often preceded by chills. Sometimes fever accompanies the disease. Vomiting and diarrhœa occur with hæmoglobinuria. Pain in the loins is sometimes complained of. The paroxysm may last a day or two, or two or three paroxysms may occur in the course of twenty-four hours.

Albumose (proteoses, propeptone, or Meissner's peptone). Formerly the reactions which determine the presence of the albumoses were thought to indicate the presence of peptone. The latter substance is extremely rare. Recent chemical investigations show that that which was called peptonuria is truly albumosuria. Albumose has been found in the urine in osteomalacia and diseases of the medulla of bone and in myxœdema. When persistent, it is in all probability due to multiple tumors of the bones or to myxœdema. The albumosuria may be considered as primary. Transi-

tory albumosuria is found in pneumonia, deep-seated suppuration, meningitis, and in dermatitis, intestinal ulcer, measles, scarlatina, and mental diseases. Its frequent occurrence renders its presence of not much diagnostic value. According to von Jaksch, its presence may indicate that a suppurative process exists. In the diagnosis of epidemic cerebrospinal from the tubercular meningitis, transitory albumosuria points to the former if no ulcerative tuberculous process exists elsewhere. Urine containing it does not respond, at first, to the heat and nitric acid test, but on cooling a precipitate forms which responds to the *biuret test*. (In this test the urine is first treated with about one-half its volume of sodium hydroxide solution, and then a 1 per cent. solution of cupric sulphate is added drop by drop. If albumose is present, the resulting cupric hydroxide is dissolved, and the fluid becomes of a violet-red color.) The probability of the presence of albumose is strengthened if a turbidity occurs with the acetic acid and potassium ferrocyanide test (acetic acid, specific gravity 1064, to which a few drops of a 10 per cent. solution of potassium ferrocyanide have been added), and also with the biuret test applied directly to the urine itself. Albumin also responds to this test.

The best test for albumoses is that of Hofmeister, modified by Sal-kowski. 20 to 50 c.c. of urine are acidified with acetic acid and then added to an equal quantity of a saturated solution of common salt, boiled and filtered. In this manner the urine is freed from albumin; the albumin remaining as a filtrate while the albumose is redissolved. The filtered fluid containing the albumose is placed in a beaker and a few drops of HCl added. A solution of phosphotungstic acid is added and the precipitate consolidated by heat into a coherent mass. Then pour off the supernatant fluid; wash the precipitate with water and dissolve in a solution of soda (specific gravity 1.16), which is added drop by drop until dissolved. If the solution is blue, it is to be gently heated, to decolorize. A few drops of a 1 per cent. solution of copper sulphate are added to the soda solution. If a red or violet color, the *biuret reaction*, results, albumose is present.

The late N. A. Randolph suggested the following test, which is given by Tyson: To 5 c.c. of urine, which must be cold and faintly acid, add 2 drops of a saturated solution of potassium iodide and then 3 or 4 drops of Millon's reagent. If albumoses or bile-acids are present, a yellow precipitate falls. If the yellow precipitate does not respond to the test for bile-acids, it is due to albumose.

Sugar (glucose). Next to albumin, sugar is the most important abnormal constituent of the urine. It is not present in normal urines in quantities that can be detected by ordinary clinical methods. The best methods for its detection are Fehling's test and the fermentation test.

Fehling's Test. Fehling's solution is prepared by dissolving 34.652 grammes of pure crystallized cupric sulphate in about 200 c.c. of water. About 173 grammes of sodic potassium tartrate (Rochelle salt) are dissolved in about 480 c.c. of sodium hydroxide solution of 1.14 specific gravity. The cupric sulphate solution is added slowly to the sodic potassium tartrate solution, stirring constantly until all of the cupric sulphate solution has been added. The bluish-white precipitate of cupric

hydroxide which first forms will, on stirring the liquid, be completely dissolved. The blue liquid is then diluted with water to exactly 1000 c.c. 1 c.c. of this solution will be reduced by 0.005 gramme of glucose. Fehling's solution is prone to undergo decomposition, and to avoid this, it is best to keep the cupric sulphate and sodio-potassium tartrate solutions in separate bottles closed with rubber stoppers. To accomplish this, the 34.652 grammes of cupric sulphate are dissolved in water and diluted to 500 c.c., and the sodio-potassium tartrate is dissolved in water and diluted to 500 c.c., and the two solutions preserved in separate bottles. The solutions, prepared in this manner, are used by mixing one volume of the cupric sulphate solution with an equal volume of the sodio-potassium tartrate solution. The resulting liquid will be Fehling's solution, and 1 c.c. of it will be equal to 0.005 gramme of glucose.

Certain precautions are necessary in the application of this test: 1. Any albumin present must be removed by boiling and filtration. 2. The Fehling solution, diluted with 4 to 5 volumes of water, must be boiled first and the urine added to it; the urine must not be boiled first and the Fehling solution added to it. Boiling the reagent first is a test of its stability; if a precipitate occurs, the solution is unfit for use. As Wormley correctly says, a precipitate is more likely to occur when the Fehling solution has been diluted with 4 or 5 times its volume of water than on boiling the undiluted solution. 3. Prolonged boiling is to be avoided. The solution is to be heated to the boiling-point and the urine then added: if no precipitate indicating sugar occurs until urine is added almost equal in volume to that of the reagent, the mixture should be again heated to the boiling-point and set aside. 4. When the earthy phosphates are abundant, it is well to get rid of them by adding a small quantity of sodium hydroxide and filtering before applying the sugar test. 5. Changes in color may occur from the presence of urea, uric acid, and extractives. These changes can be obviated, when necessary, by the method proposed by Seegen, who recommends repeated filtering through animal charcoal until the urine is rendered colorless. Fehling's test is then applied to the filtered urine.

The method of applying Fehling's test is as follows: Fehling's solution is poured to the depth of about $\frac{1}{4}$ inch into a test-tube, and diluted with 4 or 5 times its volume of water, and heated until it begins to boil; then 1 or 2 drops of the suspected urine are added. If it be ordinary diabetic urine, the mixture after an interval of a few seconds will suddenly turn to an intense opaque-yellow or reddish-brown color, and in a short time an abundant yellow or reddish-brown precipitate falls to the bottom. If, however, the quantity of sugar present be small, the suspected urine is added more freely, but not beyond a volume equal to that of the diluted Fehling's solution employed. In this latter case it is necessary to raise the mixture once more to the boiling-point. It is then allowed to cool slowly. If no cuprous oxide is thrown down when the liquid becomes cold, then the urine may be pronounced sugar-free.

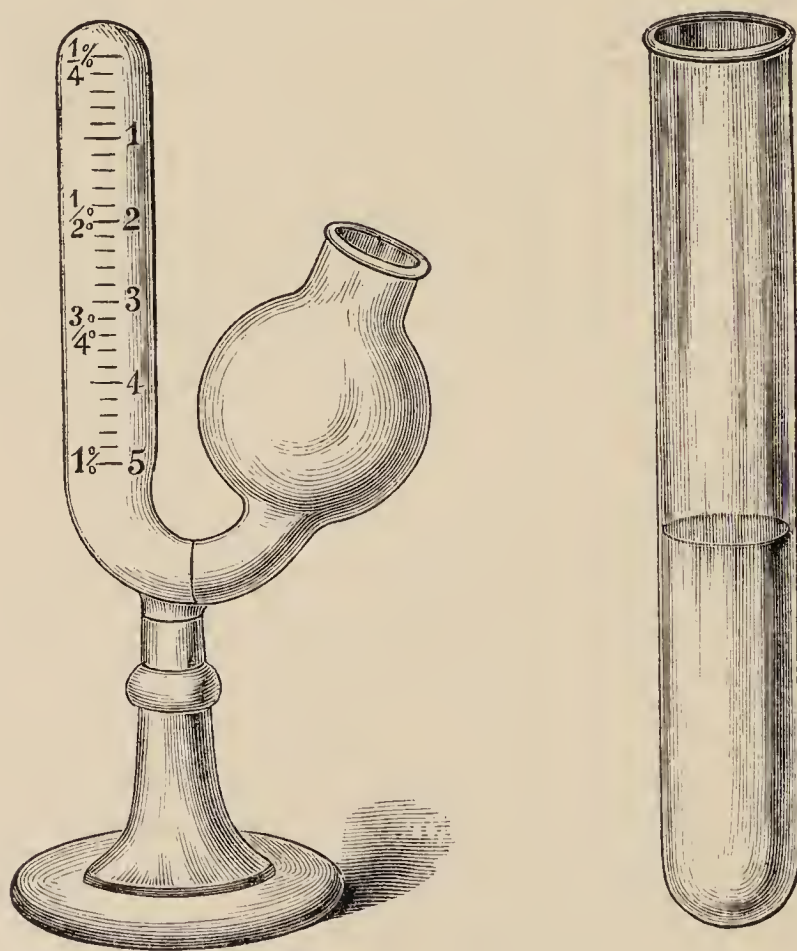
Sir William Roberts has recently pointed out the value of repeated filtration through animal charcoal of urine which reacts doubtfully to the test for sugar; by this filtration the urates, uric acid, and other normal

constituents of the urine, which have more or less power of reducing Fehling's solution, are removed, while the sugar passes through and is found in undiminished quantity in the filtrate.

The test is made as follows: A test-tube is charged with Fehling's solution to the depth of about $\frac{1}{4}$ inch, diluted with 4 or 5 times its volume of water, and brought to the boiling-point; the urine, filtered through charcoal, is added to the depth of about 2 inches, and the two fluids mixed. The flame of a lamp is then applied to the upper half of the column of liquid, and this is boiled for a couple of seconds. If sugar is present, the upper half loses its blue color and assumes a yellowish tinge, and the earthy phosphates which are thrown down in light flakes by the alkali of the test are tinted more or less of a golden color by the precipitation on them of the cuprous oxide.

The Fermentation Test. This is based upon the fact that sugar by fermentation with yeast breaks up into alcohol and carbon dioxide. It is a reliable but not a very delicate test for sugar. A piece of yeast-cake the size of a pea is added to a test-tube full of urine. The open end of the tube is inverted under water in a saucer or beaker. If sugar is present in amounts larger than $2\frac{1}{2}$ grains to the ounce, bubbles of carbon dioxide collect at the upper part of the tube after standing twelve hours in a temperature of about 90° F.

FIG. 227.



Einhorn's saccharimeter.

The Phenylhydrazin Test. Von Jaksch believes this test to be a very accurate one. About 2 grains of phenylhydrazin hydrochloride and about 3 grains of sodium acetate are put into a test-tube half-full of water. The contents of the tube are heated and the tube filled with the suspected urine. The tube is kept for fifteen or twenty minutes in boiling water,

PLATE XIII.



Crystals of Phenyl-glucosazone.

(Oc. 4, Obj. D.) Drawn by J. D. Z. Chase.

and then put in a vessel of cold water. When a large amount of sugar is present, a deposit of yellow needle-like crystals is visible to the naked eye ; but when only a small amount is present, the sediment must be examined under the microscope. The crystals appear singly, or in sheaves and fine radii. Yellow plates and brown balls do not indicate sugar. (Plate XIII.)

Quantitative Estimation of Sugar. Fehling's solution may be used with a burette and measured quantities of urine and reagent. Wormley recommends a method which answers very well for office-use : 1 c.c. of Fehling's solution is diluted in a large test-tube with 4 c.c. of distilled water, and boiled. 0.1 c.c. of the suspected urine is then added from a graduated pipette. Heat is then applied, the precipitate watched, and then another 0.1 c.c. added, and heat again applied. The addition of 0.1 c.c., followed by heat, is continued until it is found after subsidence that all color is removed from the diluted Fehling's solution. If in doing this 1 c.c. of urine has been added, it will have contained just 0.5 per cent. of sugar. If more than 1 c.c., it will have contained less than 0.5 per cent. If exactly 2 c.c. are used, it will have contained exactly 0.25 per cent. If 0.1 c.c. has been used, the urine will have contained 5 per cent. of sugar. If the quantity of sugar in the urine is large, the urine should first be diluted with a measured volume of water, allowance being made for this in the estimation.

When the quantity of sugar is relatively large, fermentation is the simplest and most trustworthy method. Roberts has shown that saccharine urine loses by fermentation 1 degree in density for every grain of sugar contained in an ounce of urine. For example, if the urine before fermentation had a specific gravity of 1040, and after fermentation a specific gravity of 1010, then the urine contained 30 grains of sugar to the ounce. In the application of this method, about 4 ounces of diabetic urine are put in a 12-ounce bottle, and a piece of Vienna yeast about the size of a pea is broken up and then added to it. This bottle is closed with a perforated cork to allow the CO_2 to escape, and stood aside in a warm place to ferment. Beside it is placed a tightly corked 4-ounce bottle filled with a specimen of the same urine, but without the yeast. In about twenty-four hours the fermentation will have ceased. The specific gravity of the fermented urine is then taken and also that of the unchanged urine. Every degree of loss in density represents 1 grain of sugar per ounce of urine.

Bremer's Diabetic Urine Test. Take 2 parts of eosin and 3 parts of gentian-violet ; dry, powder, and mix. Add to a test-tube of urine. In normal urine only the eosin dissolves, giving an orange-red tint to the urine ; the gentian-violet remains on the surface of the urine or falls to the bottom in solid masses, giving no coloration to the eosin-stained urine. In diabetic urine eosin dissolves in a few seconds ; the gentian-violet begins to fall from the surface, leaving a heavy trailing stain in the urine, which eventually becomes entirely violet. The majority of diabetic urines, especially the pale greenish shimmering ones, give the test. Occasionally the urine of chronic Bright's or of other diseases may react.

Williamson's Test. Diabetic urine to which has been added methylene-

blue in alkaline solution, will on gentle heating on a water-bath completely lose the blue color given by the stain.

Glycuronic acid occurs in urine after administration of chloral, butyl-chloral, nitrobenzole, camphor, curare, morphine, chloroform, fusel oil; reduces Fehling's solution, is dextrorotatory, and does not ferment. Many temporary glycosurias are probably due to this substance.

β -Hydroxybutyric, oxybutyric acid, in severe diabetic cases, usually with diacetic acid and acetone, has strong lævorotatory action, and may interfere with the polariscope test. If urine is strongly lævorotatory after fermentation, this substance is probably present.

Indican. An excess of indican in the urine is known as indicanuria. The substance is detected by several methods. Jaffe's test: Equal volumes of hydrochloric acid and urine are mixed. By means of a glass pipette a solution of sodium hypochlorite is dropped into the fluid. An indigo-blue color is produced if indican be present. The hypochlorite must not be added in excess. A quantitative determination is made by the colorimetric process of Salkowski. A rough analysis is first made to determine the quantity of calcium hypochlorite which causes the greatest amount of indigo to unite with it. If the urine contains much indican, a small portion, as 2.5 to 5 c.c., is diluted with water to 10 c.c.; if there is but little indican, 10 c.c. of the urine are used without dilution. An equal quantity of hydrochloric acid is added. To this the amount of hypochlorite solution with which, in the first test, indigo combined in the greatest amount is added. Then the liquid is neutralized with sodium hydroxide; and sufficient sodium carbonate is added to make it alkaline. The indigo-blue is thus precipitated and collected on a filter. The precipitate is repeatedly washed with water until the alkaline reaction disappears. The filtrate is dried and extracted by heating with chloroform until the latter no longer assumes a blue color. The chloroform extract is increased to a round number of cubic centimetres by the addition of chloroform, and placed in a vessel with parallel sides. The intensity of its color is compared with that of a freshly prepared chloroform solution of indigo-blue of known strength. To one or other of these, chloroform is added until the tint of both is the same. The quantity of indigo-blue derived from the urine is determined, and its percentage calculated from the intensity of color and strength of the solution of indigo of known strength. In health 5 to 20 mgm. of indigo-blue are passed in twenty-four hours.

Indican is increased by animal diet—an increase which, under other circumstances, is pathological. Its presence is a sign of intestinal putrefaction. It may accompany decomposition of albumin in cavities. It is present in empyema and in puerperal peritonitis. By detection of its presence in these diseases cavities due to pus may be distinguished from those due to other causes. Indican is increased in acute diarrhoea and in intestinal tuberculosis. Von Jaksch states that large quantities of indican in the urine imply that abundant albuminous putrefaction or putrid supuration is in progress in the system. It must not be forgotten that indicanuria will often arise in simple constipation.

Bile-pigments and Bile-acids. Bile-pigment, or bilirubin, occurs in

the urine in cases of hepatogenic and hæmatogenic jaundice, and in portal thrombosis.

Gmelin's test and its modifications are the ones usually employed: a small quantity of nitric acid, to which some nitrous acid has been added, is put in a test-tube and then gently overlaid with urine: if bile-pigment is present, a series of colors appear at the junction of the two fluids—green, blue, violet, and yellow. A green color (biliverdin) must be present to prove the existence of bile-pigment.

The same test may be applied by placing a few drops of the acid upon one side of a plate and the urine on the other, and then allowing the two to run together: the play of colors takes place, as before, at the line of junction of the acids and urine.

Rosenbach's modification is an improvement: about 200 c.c. of urine are allowed to flow through pure white filter-paper, and then a drop of nitric acid is placed upon the paper saturated with the urine: the colors appear as before described.

A very simple test consists in allowing a few drops of nitric acid to fall into a test-tube full of urine: if bile-pigment is present, a green color appears at the line of junction of the two fluids. This test may fail, however, if only small quantities of bile-pigment are present.

The tests for bile-acids are either too elaborate or too unsatisfactory for clinical use.

Pus. Pus is found in the urine whenever there is suppuration or a catarrhal condition of the genito-urinary tract. Hence it occurs in abscess of the kidney, pyonephrosis, pyelitis, tuberculosis, cystitis, gonorrhœa, leucorrhœa, etc. It is relatively common in women, from a catarrhal condition of the vulva and vaginal mucous membrane, and is therefore of less significance than in men. Urine containing much pus is slightly albuminous; but frequently pus-cells are found in urine which gives no reaction for albumin.

The chemical test for pus is its conversion into a tenacious (gelatinous), glairy mass by boiling with caustic potash.

Acetonuria. An excess of acetone occurs in the following diseases: (1) in diabetes; (2) in cancer independent of starvation; (3) in starvation; (4) in certain psychoses; (5) in auto-intoxications; (6) in derangement of digestion; (7) in fevers. In diabetes acetone indicates an advanced stage of the disease. Lieben's test for acetone is as follows: to several cubic centimetres of distilled urine a few drops of iodo-potassic iodide solution and sodium hydroxide are added: if acetone is in excess, precipitation of iodoform takes place, which may be recognized by its odor.

Diaceturia. Diacetic acid is found in the urine in diabetes, in fevers, and in auto-intoxications. It is common with children in fever. It is of grave significance in the urine of adults. Coma usually follows its occurrence in the urine in fevers and in diabetes. Test: a concentrated solution of ferric chloride is cautiously added to the urine: if a precipitate be formed, it should be removed by filtration and more chloride added to the filtrate. If diacetic acid be present, the liquid will become claret-red in color.

Hæmatoporphyrinuria. This is a rare constituent of the urine derived from the blood. It is said to be a form of hæmatin freed from iron. Nakarai thinks that the occurrence of hæmatoporphyrinuria is constant in lead-poisoning, and occurs with some degree of frequency in intestinal hemorrhage.

Alkaptonuria. The substance in the urine which has been identified as alkapton is also known as pyrocatechin,¹ protocatechuic acid,² urrhodinic acid,³ glycosuric acid,⁴ uroleucinic and unoxanthinic acids,⁵ and homogentisinic acid.⁶ It reduces copper, as does glucose, and its occurrence is of interest because the presence of the substance has led to the diagnosis of glycosuria in many instances, in consequence of which persons have been refused life insurance. The urine containing this substance deepens in color on exposure to air. It is of a peculiar aromatic odor, and reduces cupric salts rapidly. There is, however, no reaction to the fermentation test, to Böttger's bismuth test, or to phenylhydrazin, and no deviation of polarized light. The urine does not contain bile-pigment. It is of normal specific gravity, and becomes very dark on the addition of an alkali, or of a temporarily bluish-green color with ferric chloride. Ammonio-nitrite of silver is instantaneously reduced when added to the urine, with a deposit of metallic silver.

Alkaptonuria is usually congenital. Several members of the same family may have it. No symptoms attend the condition.

NOTE.—*Serum-globulin* is converted into a coagulated proteid when heat is applied or concentrated nitric acid added to a solution. Globulin is soluble in dilute salt solutions. If urine rich in globulin is added drop by drop to a large volume of distilled water, the globulin is precipitated as the percentage of salt is reduced by dilution. Globulin is also precipitated by dialysis. If a portion of the urine containing globulin is saturated with magnesium sulphate or half-saturated with ammonium sulphate, globulin is precipitated.

Hills⁷ describes the method as follows: "25 to 50 c.c. of the urine are made neutral or slightly alkaline with ammonium hydroxide, and the precipitated phosphates removed by filtration. An equal volume of a saturated solution of ammonium sulphate is then added, the mixture shaken and allowed to stand for some time, and finally filtered. The precipitate is washed with a half-saturated solution of ammonium sulphate for the removal of the last traces of albumin, and the filtrate and precipitate tested for albumin and globulin respectively, as previously described. The formation of a precipitate upon the addition of either magnesium or ammonium sulphate is not in itself evidence of the presence of globulin.

¹ Ebstein and Müller, Virchow's Archiv, Bd. lxx. S. 394.

² Smith, Dublin Journal of Medical Science, 1882, vol. i. p. 465.

³ Kirk, British Medical Journal, London, 1886, vol. ii. p. 1017.

⁴ Marshall, Medical News, Philadelphia, 1887, p. 35.

⁵ Kirk, British Medical Journal, London, 1888, vol. ii. p. 232.

⁶ Baumann and Wolkow, Zeitschr. f. physiol. Chem., Strassburg, Bd. xv. S. 228.

⁷ Boston Medical and Surgical Journal, 1899, vol. cxli. No. 6.

Microscopical Examination of the Urine.

Microscopical examination of the urine is chiefly concerned with the sediments, and these are conveniently divided into the organized and unorganized.

The *organized deposits* in the urine are blood, pus, mucus, epithelium, casts, spermatozoa, micro-organisms, cancerous and tuberculous matter, and entozoa.

FIG. 228.



Extraneous matters found in urine: *a*, cotton-fibres; *b*, flax-fibres; *c*, hairs; *d*, air-bubbles; *e*, oil-globules; *f*, wheat-starch; *g*, potato-starch; *h*, rice-starch granules; *i, i, i*, vegetable tissue; *k*, muscular tissue; *l*, feathers.

The *unorganized deposits* are uric acid and its compounds, oxalate and carbonate of lime, phosphates, leucin and tyrosin, cystin, and cholesterolin.

Normal urine forms a slight sediment, consisting of epithelium from different parts of the genito-urinary tract, principally from the bladder in males and from the vagina and bladder in females. There are also crystals of the different urinary salts, sometimes mucus, and a few white blood-cells; and if the urine has stood a while, especially if it is alkaline,

more or less bacteria. It may accidentally contain extraneous matters derived from the vessel which contains it or from the air. (Fig. 228.)

The centrifugal machine has now become an important adjunct to the rapid and accurate microscopical examination of the urine. There are numerous varieties to be secured at the instrument-stores, some of which are devised solely for urinary examination, while others have additional apparatus for examination of the blood and sputum. The majority of them are revolved by hand. Electricity can be readily applied to any of them and labor be saved by such a device. The advantages of centrifugal force over the older gravity method employed in microscopical examination are marked. Some few of them can be briefly outlined :

1. Centrifugalization secures complete, rapid, and concentrated sedimentation. It is therefore best suited to microscopical diagnosis.

2. Casts or other organic material, if present, can be studied carefully before they are macerated or partially destroyed by bacteria or changed by the deposition of amorphous or crystalline material. This is a most important aid to correct diagnosis.

3. Crystals, if present at the time of urination, can be discovered and differentiated from those that normally crystallize out after some hours.

Certain bodies—hyaline casts, for instance—because of their rather light specific gravity, do not settle on simple standing of the urine, and thus escape detection. These with all other substances are thrown down with the centrifugal machine.

5. Bacteria are discovered with greater ease, especially the tubercle bacillus.

The method commonly used for the examination of the urinary sediment is as follows: The urine for examination (the chemical analysis having previously been made) is decanted until there remains but a small amount in the bottle, which amount contains any sediment already formed and heavier organic materials. This is then poured into one of the tubes of the centrifugal machine to within one-half inch of the top; if but one specimen of urine is to be examined, fill both tubes with specimens from the same urine. If there is not sufficient urine to do this, fill the remaining tube or tubes with water. It is well to mark the external metal shields of the tubes with a figure, say 1 and 2, or *a* and *b*, so that the urines, if different specimens, may not become confused. The tubes are then rapidly revolved for three minutes, removed from the machine and a few drops of the sediment withdrawn with a pipette and placed upon the slide for examination under the microscope. It is necessary to remember that care must be exercised in removing this sediment from the tube. The straight glass pipette without a pointed end seems to give the best results in securing the sediment. The finger is placed upon one end, the pipette inserted to the bottom of the tube, and the finger elevated just enough to secure a few drops of the sediment that has been cast down by centrifugalization. The same object is attained if the finger is entirely removed as soon as the point of the tube reaches the bottom of the conical glass; but in that case more than the lowest layers of the sediment are sucked up, and hence all but a few drops should be allowed

to flow out when the tube is removed from the urine. In this way the drops reserved for the microscopical examination will contain the sediment from the very bottom of the glass. In this sediment, in pale urines free from much urates, phosphates, and pus, the casts will be found, if any are present in the urine. It is most important to examine the bottom layers of the sediment when the latter is scanty, or when phosphates or urates have begun to precipitate after the urine has been standing some time. If the urine is already cloudy with phosphates, urates, or pus, when it is put aside to settle, any casts that may be present will be carried down with the heavier sediment, and will be found intimately mixed with it, or even on top of the other sediment. The few drops reserved for microscopical examination are now deposited on several slides without a cover-glass, and examined carefully for organic and inorganic constituents.

If the urine contains but the normal amount of mucous cloud, a very small whitish sediment or cloud is found at the bottom of the tube. If calcium oxalate is present, a small filmy, whitish sediment is seen. The sediment of amorphous urates is pinkish, fawn, or salmon color. Uric acid appears as a "brick-dust" sediment. Pus produces a heavy yellowish sediment; phosphates a heavy white sediment, which is sometimes yellowish white from admixture with leucocytes. Blood in small quantities produces a rather characteristic brownish deposit. Large amounts of blood appear as reddish coagula at the bottom of the tube.

With some of the centrifugal machines the various urinary salts and the amount of albumin present can readily be estimated. Such instruments are provided with graduated tubes, into which the urine and the necessary reagents are put and the resulting precipitate rapidly cast down.

In this manner Purdy estimates the chlorides, sulphates, and phosphates, and also the amount of albumin most satisfactorily. It is questionable, however, whether the estimation of the salts is accurate.

If the centrifugal machine can not be employed, proceed as follows: 6 or 8 ounces of the urine to be examined should be allowed to settle in a bottle as soon after being passed as possible. The bottle should be tightly corked, because urine exposed to the air decomposes very quickly; it should be sent to the laboratory as soon as possible after being passed, in order that an examination may be made before fermentative changes spoil it for trustworthy analysis. After standing twelve, or preferably twenty-four hours, nearly all of the solid matter will have collected at the bottom of the bottle. The supernatant clear fluid can now be poured off, and the lower portion of the urine and the sediment poured into a conical subsiding-glass. If the urine is febrile, there may be by this time a large deposit of amorphous urates, which will obscure the search for casts; they may be dissolved by gentle heating without destroying the casts, and the clear urine again allowed to settle for a few hours. So, too, if phosphates are abundant, they should be gotten rid of by gentle heating, and by acidulation with 2 or 3 drops of dilute acetic acid. After the urine in the conical subsiding-glass, which will now amount to an ounce or two, has stood for a few hours, any casts that may be present will have fallen to the bottom. If the urine is highly concentrated (1030

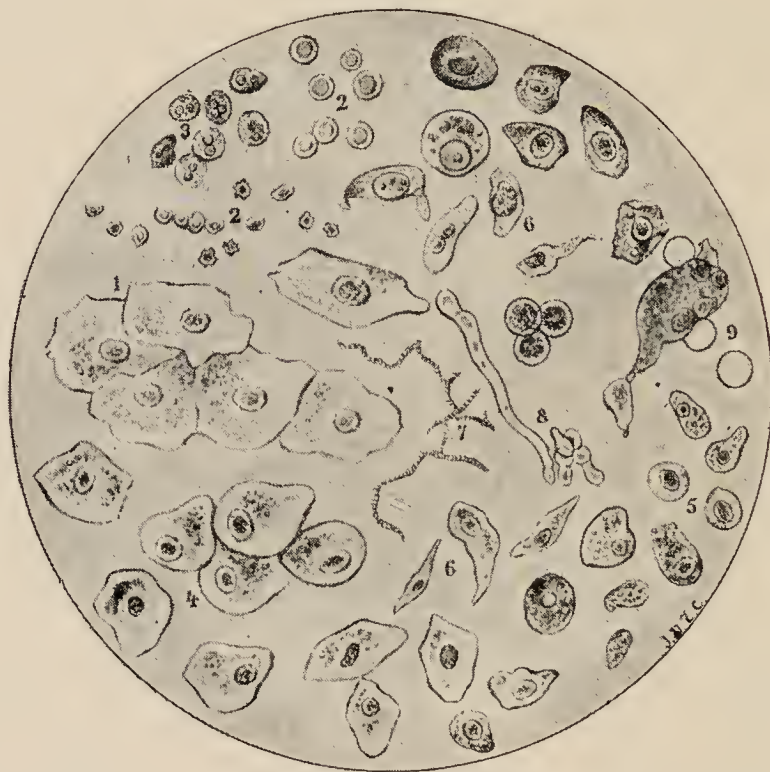
or more), epithelium, blood, and casts will be suspended longer; hence it may be well to dilute the urine before allowing it to settle. A specimen of the sediment is then transferred to a slide for microscopical examination, as already described.

All the pipettes used in examining urine must be kept clean. They should be allowed to stand in water which is frequently changed, and carefully rinsed in running water before being used.

Organized Sediments.

Blood. If the blood comes from the kidney, it is usually intimately mixed with the urine, which remains of a red or reddish-brown color, and contains possibly tube-casts and renal epithelium. The blood-cells appear singly, have frequently lost their hæmoglobin, and hence look like pale-yellow disks. (See Fig. 229.)

FIG. 229.



Cellular elements from the urine: 1, squamous epithelium; 2, red blood-corpuscles; 3, polynuclear leucocytes; 4, transitional cells; 5, epithelium from the kidneys; 6, epithelium from the bladder; 7, *Micrococcus aureæ*; 8, yeast-fungi.

Sometimes blood coagulates in the ureters, and long, cylindrical plugs are passed, causing symptoms resembling those of renal colic. When blood comes from the bladder or neck of the bladder (fissure), there are frequent micturition, acute pain and tenesmus, and the blood is not intimately mixed with the urine. When the hemorrhage is from the neck of the bladder, it often occurs as a few drops of blood at the end of micturition, accompanied with great pain and a sense of faintness. Intermittent hæmaturia, according to von Jaksch, points directly to calculus or tumor of the bladder.

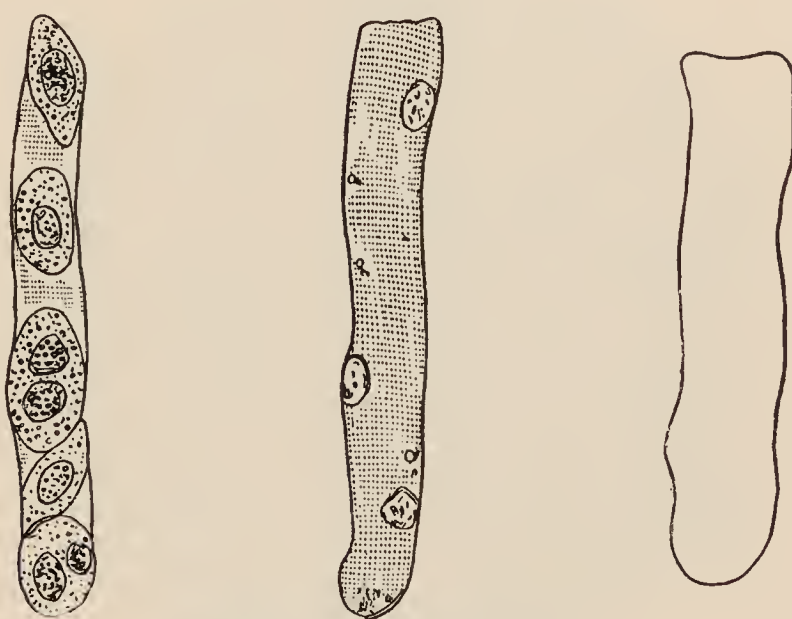
Blood-cells when unaltered are unmistakable on account of their well-known biconcave appearance. When they have lost their coloring-matter they appear as circular, very pale disks, with extremely faint outline and feeble refractive power. Absence of a nucleus serves to distinguish them

from yeast-spores, and the latter, moreover, are often oval in shape. They are less likely to be confounded with the ovoid and circular shapes of calcium oxalate crystals, because the latter are not common, and can be seen usually in their more common forms as octahedra and dumb-bells in the same urine.

Pus. The sources of pus in the urine have been mentioned. The pus-corpuscle is an opaque, spherical, granular cell, usually somewhat larger than blood-cells. In concentrated urines the pus-cell is small; in dilute urine, or urine to which water has been added, it swells sometimes to twice its original size. At the same time it becomes less granular, and two, three, or four nuclei may appear. The addition of acetic acid also causes it to swell, and brings out the nuclei more distinctly and rapidly. Sometimes the pus-cells are discrete, sometimes in dense clumps, and sometimes nothing but a dense mass of pus-cells appears in the field of the microscope.

The source of the leucocytes must be a matter of inference from the general characters of the urine. If red blood-cells are also present, the probability of finding white blood-cells is increased, but pus-cells are not necessarily excluded. So, too, if much mucus be present in the urine, the doubtful cell may be a mucus-corpuscle. Some clue to the source of the pus can be obtained from the urine itself. Urine containing pus from the kidney is usually acid, whereas in cystitis it is alkaline, and almost always contains phosphates, mucus, and abundant bacteria. An exception to this is found in the cystitis of certain general infections. Thus typhoid cystitis and cystitis due to *Bacillus coli communis* are regularly associated with acid urine. Again, pus from the kidney or kidney pelvis is apt to vary greatly in amounts or be discharged intermittently; and the urine when filtered free from pus-cells is usually still albuminous. Renal epithelium and casts may also be found.

FIG. 230.



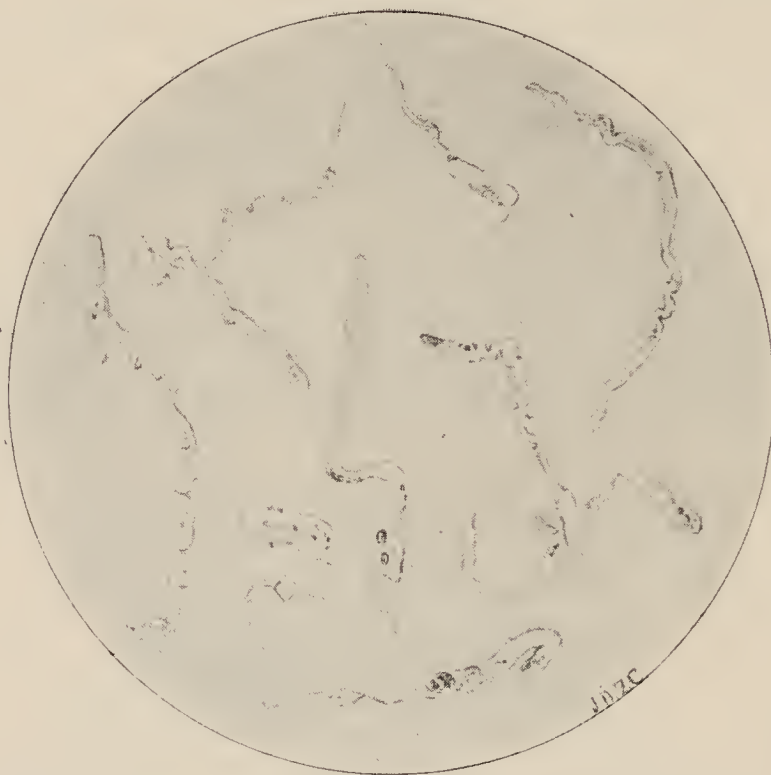
Epithelial and hyaline casts.

Casts. Casts are the most important of the urinary deposits. They vary greatly in number and size.

Casts may be numerous, so that nearly every field contains a dozen or more; or they may be very few, not more than one or two being found

on a slide. The best routine method for microscopical examination is as follows: place a few drops of the urinary sediment upon the slide; spread the drops in a thin layer; use no cover-glass; *examine with the low power*—a diameter of 50—with a small amount of light; the whole slide can

FIG. 231.



Hyaline casts and cylindroids in hypostatic congestion of kidney. Low power.

be carefully searched in three minutes, and the casts discovered can be minutely studied with the higher power. When but few casts are present, several slides can be rapidly examined with the low power, and an accurate estimation of the number made.

FIG. 232.



Hyaline casts from a case of acute nephritis: 1, plain hyaline cast; 2, granular deposit on hyaline cast; 3, cellular deposit (blood and epithelium).

Tube-casts usually indicate acute or chronic nephritis; but they are *sometimes* found in cases of renal calculi; in icterus, usually without

PLATE XIII-a.

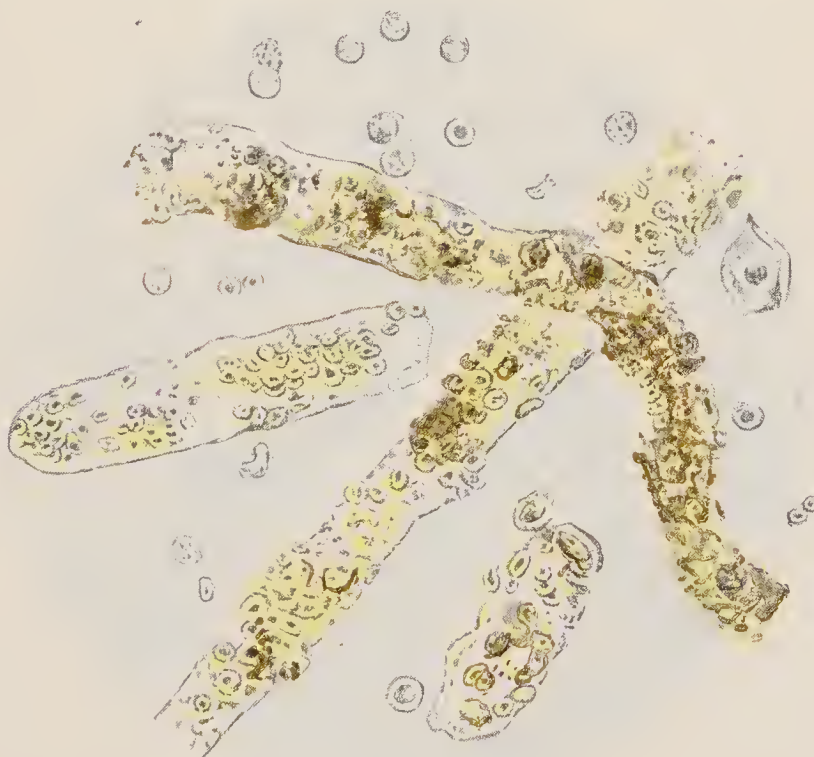
FIG. 1.



1. Hyaline Casts with Granular Matter and Epithelial Cells deposited upon them. 2. Amyloid (waxy) Cast.

(Oc. 4, Ob. D.) Drawn by J. D. Z. Chase.

FIG. 2.



Blood-Casts from Case of Acute Nephritis.

(Oc. 4, Ob. D.) Drawn by J. D. Z. Chase.

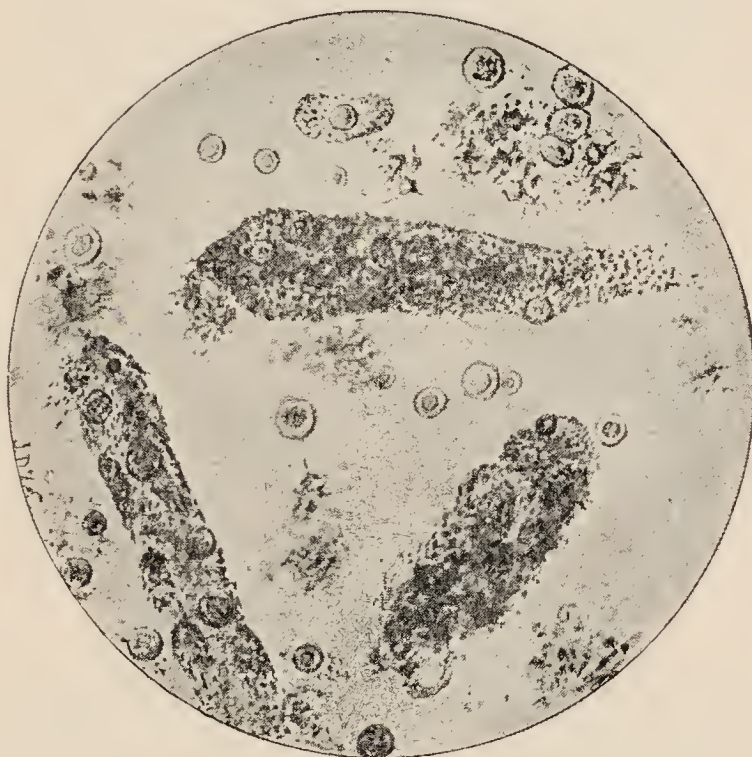
albuminuria ; in diabetes, and sometimes in secondary congestion of the kidney.

Several varieties of casts are found.

1. **Hyaline casts**, as their name implies, are clear, translucent bodies, which refract light so slightly that they are easily overlooked. They have well-defined margins, the ends being frequently rounded ; they are rarely very long, and are straight or but slightly bent. They are rarely equally translucent throughout ; at some part more or less granulation will generally be found. They vary in diameter from that of a white blood-cell to 6 or 8 times as large. They can be stained, and so rendered more distinct, by allowing a drop of gentian-violet solution to flow in under the edge of the cover-glass. (Figs. 231 and 232.)

2. **Granular casts** are hyaline casts which appear granular either from some deposit on their surface or from a granular change of the cast itself.

FIG. 233.



Granular casts.

When the granulation does not interfere with the translucency, the casts are described as "pale" or "slightly" granular ; and when they become very dark, so as to resemble closely a blood-cast, they are called "dark" or "opaque" granular casts. (Plate XIII.—A, Figs. 1, 1 ; and Fig. 233.)

3. **Waxy casts** appear to the eye to be more solid in structure than the hyaline casts ; they also appear more cylindrical in form, are more or less yellow in color, and are apt to be larger than hyaline casts. (Plate XIII.—A, Figs. 1, 2.)

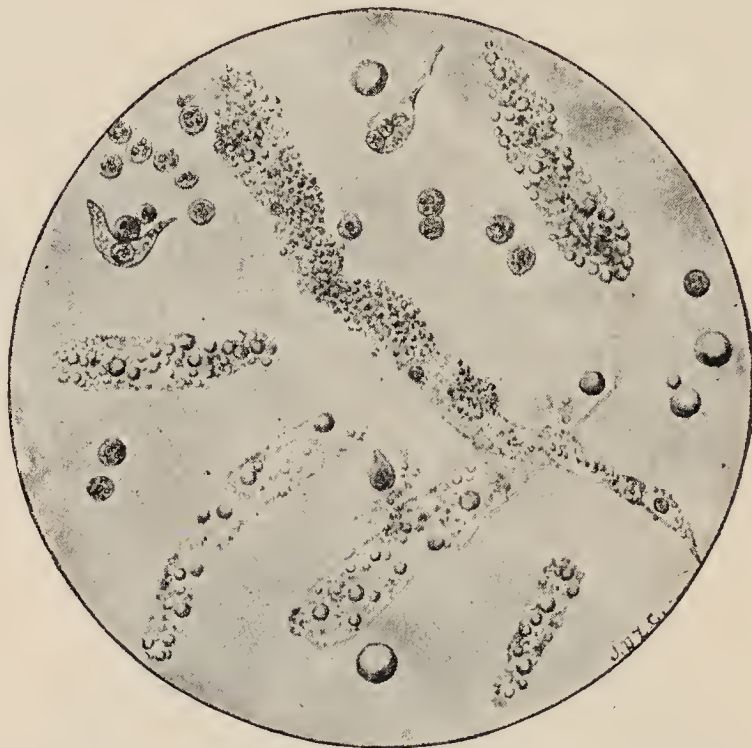
4. **Fatty casts** are hyaline or faintly granular casts on which are deposited, in spots, minute oil-drops. If the oil-drops are very abundant, they are sometimes called "oil-casts." (Fig. 234.)

5. **Blood-casts** are either made up of a mass of blood-cells pressed into a cylindrical shape, or more frequently they are hyaline casts studded with blood-cells. (Plate XIII.—A, Fig. 2.)

6. **Epithelial casts** sometimes seem to be composed entirely of epithelial

cells closely packed. Such casts are relatively rare, and very beautiful. Ordinarily, just as in the case of blood-casts, an epithelial cast consists of a hyaline cast more or less covered with renal epithelium. (Plate XIII.—A, Figs. 1, 1 ; and Fig. 230.)

FIG. 234.

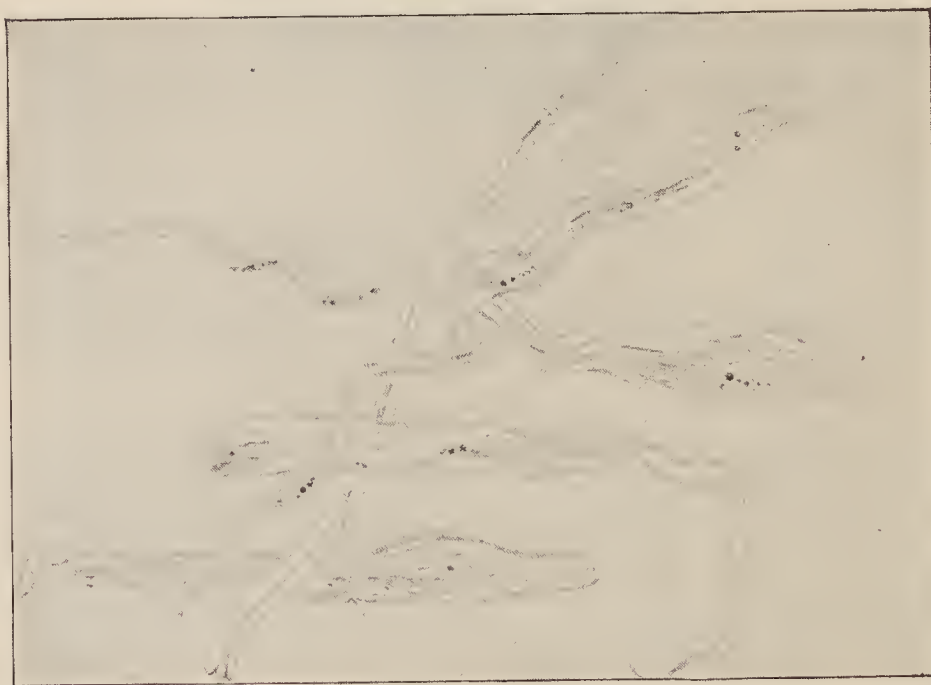


Fatty casts from a case of chronic parenchymatous nephritis.

7. **Pus-casts** are usually described as more or less regularly occurring in purulent kidney affections.

8. **Cylindroids** are very common. In general appearance they resemble hyaline casts ; but they are apt to be much longer, bent, twisted, or split, and to have a striated or finely ribbed appearance on close examination.

FIG. 235.



Cylindroids.

Moreover, the diameter of the cast frequently varies greatly at different points ; sometimes it appears constricted in several places, and in other cases one end tapers into a thread. Often cylindroids consist of fine, narrow, ribbon-like threads. (Figs. 231 and 235.)

Spermatozoa. Spermatozoa are easily recognized by their tadpole shape and by the vibratile motion of their long, delicate tails. They are found in the urine of both sexes after sexual intercourse. (Figs. 236 and 237.)

FIG. 236.



Human semen: *a*, spermatozoa; *b*, cylindrical epithelium; *c*, bodies enclosing lecithin granules; *d*, squamous epithelium from the urethra; *d'*, testicle-cells; *e*, amyloid corpuscles; *f*, spermatic crystals; *g*, hyaline globules. (VON JAKSCH.)

Many continent men have occasionally nocturnal emissions, accompanied by erections and erotic sensations. These can not be looked upon as abnormal, and they are compatible with robust health. There are other persons, neurotic, anæmic, and generally constipated in habit, who have emissions at night two or three times a week, of which they are

FIG. 237.



Spermatozoa from urine. (Original.)

unconscious until they awake and find themselves wet. Semen may also be lost during micturition and defecation, especially when much straining is required. Such a condition (spermatorrhœa) is abnormal. It is due to general nervous and muscular relaxation, associated with nervous dyspepsia and anæmia, and aggravated by sedentary life, constipation, and the reading of salacious literature or the cultivation of erotic thoughts.

In young men, it sometimes follows habits of masturbation, which have been broken up but have left behind a hyperæsthetic condition of the prostatic portion of the urethra, with or without dilatation of the orifices of the ejaculatory ducts ; or a stricture of gonorrhœal origin may be its cause. Students and over-worked and over-strained business and professional men are the ones most frequently affected.

However caused, the condition is apt to beget a most distressing state of despondency, in which the patient imagines all possible ills, and is liable to drift into a hysterical, melancholic, even suicidal frame of mind, and so falls a victim to quacks.

Epithelium. Epithelium from the kidney, bladder, and genito-urinary passages occurs in the urine. Epithelial deposits in male urine are very scanty unless there is some disease of the kidney or bladder, or a catarrhal condition of the prostatic urethra, such as is left from an old gonorrhœa. On the other hand, considerable epithelium may be normally present in the urine of women, being derived principally from the vagina and bladder.

Vaginal epithelium consists of large flat pavement-cells, and is readily distinguished.

The type of epithelium of the kidney, kidney pelvis, ureter, and bladder is the same, and it is not possible to distinguish with certainty the cells which come from each. If the cells are scanty, von Jaksch thinks they come from the ureter. He has found them in moderate quantities and superimposed upon one another.

Renal cells closely resemble the oval polygonal cells from the deeper layers of the bladder, but they have a relatively larger nucleus. (See Fig. 229.)

Fat. Oil is found in the urine in fatty degeneration of the kidney and its epithelium, and occasionally in the urine of those who are taking cod-liver oil, and in calculous diseases of the pancreas. Tyson suggests that it may come from cystic cheesy degeneration of the kidney. It is also found in chronic nephritis, in phosphorus-poisoning, and in diabetes mellitus, as well as in chyluria. The urine is turbid, but clears when agitated with ether. The fat may be separated by a sedimentator, and can be recognized by its refractive properties.

Staining for Fat. Reeder recommends Sudan-3 for staining human secretions and excretions to determine the presence of fat. Large fat-droplets take a bright-red, and small droplets a yellow or orange color. Fat can thus be demonstrated in the blood in lipæmia, lipuria, and chyluria. By this method fat can be demonstrated in the stomach contents and in the feces of adults with jaundice. A saturated solution of Sudan-3 in 96 per cent. alcohol is employed. Equal parts of this solution and 96 per cent. alcohol are added to the urine. In urinary sediments the fat-droplets in casts stain a scarlet red.

Chyle. Chyluria is a more or less milky condition of the urine, due to the presence of fat, which probably gains entrance to some part of the urinary tract by rupture of the lymphatic vessels. A case has been reported by Saundby in which a young unmarried girl being pregnant, compressed her abdomen so much, in order to conceal her condition, that

œdema of the legs, thighs, vulva, and lower parts of the abdomen resulted. After confinement the urine became milky, and remained so for many days. It contained fatty matters and cholesterin, but no albumin or sugar.

Fat and albumin appear at the same time in *parasitic chyluria*, which is due to *Filaria sanguinis hominis*, whose embryos obstruct the lymphatics. They recur at long intervals. Red and white blood-corpuscles are also found in small amounts. The urine coagulates on standing or gelatinizes.

Entozoa. The most common is the *echinococcus* or *hydatid*. When this infects the kidney or urinary vessels, hooklets and even cysts have been passed in the urine. The disease is extremely rare in this country.

Filaria sanguinis hominis, which causes parasitic chyluria, is occasionally found in the urine. (See *Filaria*.)

Bilharzia hæmatobia sometimes lodges in the urinary tract and causes hæmaturia. It is peculiar to Egypt.

Distoma hæmatobium. Common in Egypt and Abyssinia. Eggs collect in great masses in the urinary passages, and lead to inflammation, ulcers, stenosis, etc. Eggs found in the urine alone make the diagnosis possible.

Strongylus Gigas. Very rare. Symptoms of pyelitis. (The parasite is of the size of an earth-worm.)

Intestinal worms may creep into the bladder through fistulous or other openings, and be discharged through the urethra.

Micro-organisms. Normal urine contains no micro-organisms at the time it is voided. As the result of exposure to air, however, they may develop in great abundance. The non-pathogenic organisms found are classed as mould-fungi (hyphomycetes), yeast-fungi (blastomycetes), and fission-fungi (schizomycetes).

Mould-fungi, according to von Jaksch, are found rarely in foul normal urine. Yeast-fungi are also rare in normal urine. Fission-fungi are found in urine which is undergoing ammoniacal decomposition.

Sarcinæ, usually smaller than those of the stomach, are occasionally met with, especially, according to Roberts, when there is some disorder of the urinary organs, renal pains, painful micturition, cystitis, etc.

FIG. 238.



Vibriones in urine. (ROBERTS.)

Under the name *bacteriuria*, Roberts and others have described cases in which the urine contained bacteria at the time of being voided. He makes four groups: (1) cases in which the presence of bacteria is associated with incipient putrefactive changes in the urine; (2) cases associated with ammoniacal fermentation of the urine; (3) cases in which common forms of bacteria are present without decomposition of the urine; and (4) cases in which micrococcus-chains are voided in the urine.

The pathogenic organisms which are more or less closely associated with infectious diseases, as typhoid fever, septic processes, and tuberculosis, are found at times in the urine, and can be demonstrated by the proper staining-methods or cultural properties.

Dock has given an admirable account of the occurrence of the trichomonas in the genito-urinary passages. This parasite belongs to the flagellate infusoria. The prominent symptoms caused in Dock's case were painful, difficult, and frequent urination, followed by hæmaturia. The urine contained pus, epithelium of all kinds, and a number of bodies slightly larger than pus-corpuscles of a peculiar amyloid appearance—the trichomonades.

Morbid Growths. The urine very rarely contains the elements of morbid growths. Von Jaksch says he never has found the urinary findings in any way reliable in the case of tumors of the kidney. The detection of cancer cells or pigmented cells, such as occur in melanotic cancers, may confirm the diagnosis if the clinical symptoms point to cancer. Tumor-elements are most likely to be found in ulcerating tumor of the bladder.

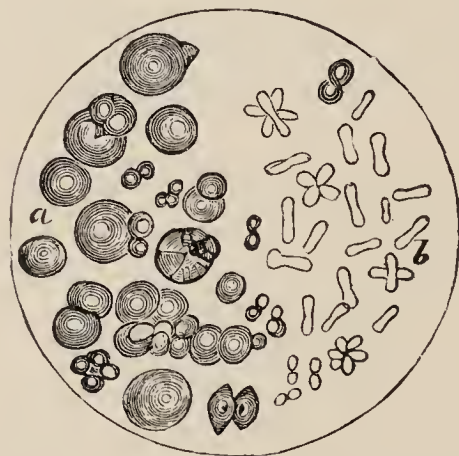
Unorganized Sediments.

Uric Acid. Uric acid is present in small quantities (8 to 10 grains a day) in normal urine. It is *increased* in febrile and wasting diseases, such as phthisis; in diseases of the liver and spleen (leukæmia), and in malarial fever, diabetes, scurvy, rhachitis, and following an attack of gout. Excessive use of milk is said to increase it. Its excretion is also increased by certain drugs—colchicum, corrosive sublimate, salicylic acid, and euonymin. It is *diminished* in anæmia, chlorosis, during a paroxysm of gout; in chronic nephritis; and by the use of certain drugs—large doses of quinine (Ranke), caffein, sodium chloride, sodium carbonate, lithia, and potassium iodide. (Plate XIII.—B, Figs. 1 and 2.)

FIG. 239.



Sodium urate: *a, a*, from a gouty concretion; *b, b*, artificially prepared by adding liquor sodæ to the amorphous urate deposit. (ROBERTS.)

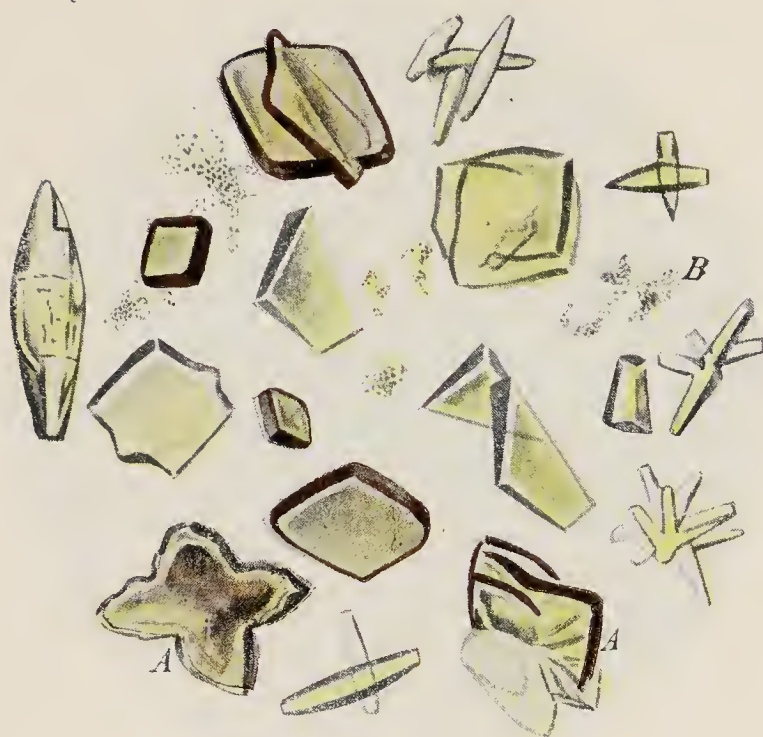


Ammonium urate spontaneously deposited: *a*, spheres and globular masses; *b*, dumb-bells, crosses, rosettes. (ROBERTS.)

According to Roberts, a deposit of uric acid occurring twelve to twenty-four hours after the urine has been passed has no pathological significance. If the deposit occurs within three or four hours after the urine has been passed, it is certainly not natural. It is frequently

PLATE XIII-b.

FIG. 1.

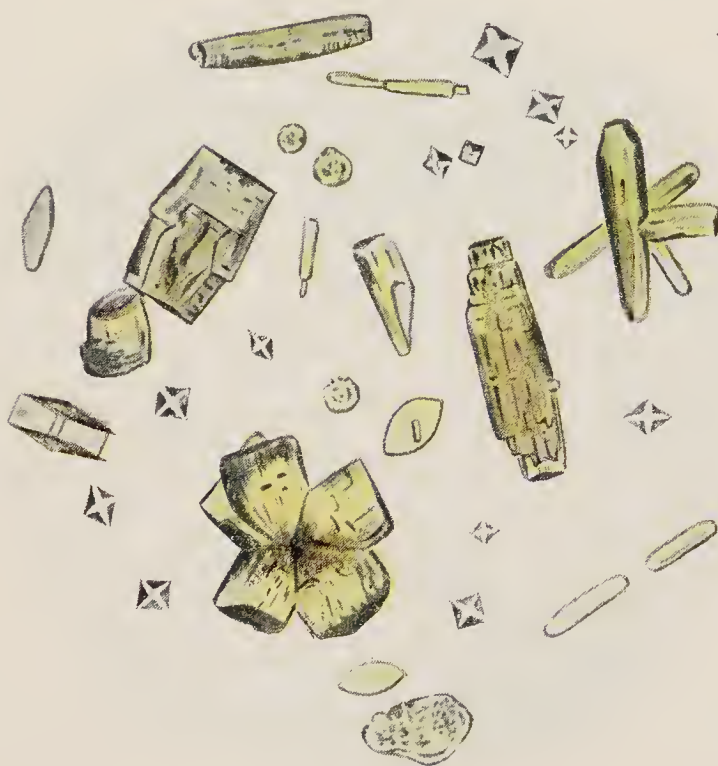


Uric Acid.

A. Common forms. *B.* Amorphous urates.

(Ob. D. and A., Oc. 4.) Drawn by J. D. Z. Chase.

FIG. 2.



Combination of Uric Acid and Calcium Oxalate.

(Oc. 4, Ob. D.) Drawn by J. D. Z. Chase.

observed in convalescence from febrile complaints, especially articular rheumatism; also in the middle periods of chronic Bright's disease, in chorea, in certain types of diabetes, and in enlargement of the spleen.

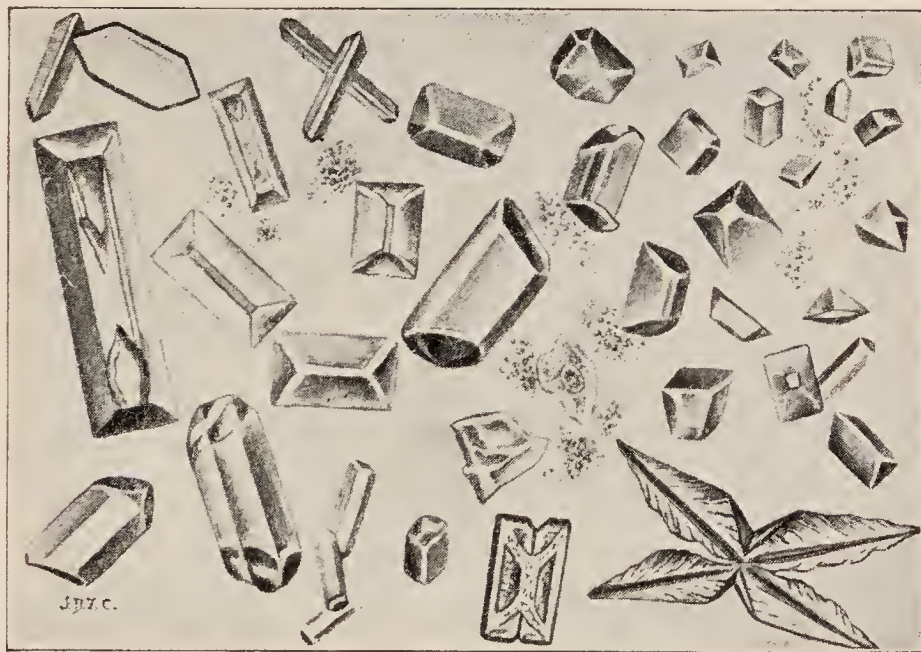
FIG. 240.



Ammonium urate. (Original.)

If, however, the uric acid is precipitated before the urine cools, or immediately afterward, it is possible that the same precipitation may occur within some part of the urinary channels, and so form a calculus.

FIG. 241.



Triple phosphates. (Original.)

Urates. Amorphous urates appear under the microscope as opaque granular particles, which dissolve upon heating, and respond to the murexide test. The deposit is more or less dense, and is sometimes arranged so as to resemble granular casts.

Sodium urate appears as spherules or globules, from which project short spines either straight or curved. It occurs most frequently in concen-

trated acid urines, such as are passed by children with acute febrile diseases. (Fig. 239.)

Ammonium urate resembles sodium urate. It is frequently associated with phosphatic deposits, and is precipitated from alkaline urines. Sometimes it appears in the shape of dumb-bells. (Figs. 239 and 240.)

Phosphates. Phosphates appear in the urine as ammonio-magnesium phosphate and as the crystalline and amorphous calcium phosphate. They are precipitated in alkaline or faintly acid urines, which produce a cloud upon being heated; the cloud is distinguished from albumin, as already pointed out, by the fact that it disappears when the urine is acidulated with acetic or nitric acid. Ammonio-magnesium phosphate is easily recognized by its rhombic prisms—"coffin-lid" shape. Other shapes are produced by modification of the primary one, chiefly by bevelling of the edges and hollowing out of the sides. These crystals are usually large, and are frequently found, together with amorphous phosphates, bladder epithelium, and pus, in cases of cystitis.

FIG. 242.



Calcium phosphate crystals. (Original.)

Amorphous phosphate of lime consists of fine granular particles much resembling amorphous urates, but distinguished from them by not disappearing upon the application of heat, but instantly dissolving when the urine is acidulated.

Crystalline phosphate of lime is a not infrequent deposit. It is found as narrow-wedged crystals, occasionally grouped in the form of stars, sheaves, or bundles, with their apices at a common centre.

According to Roberts, this deposit in quantity is an accompaniment of some grave disorder. He has found the stellar phosphates in cancer of the pylorus, once in phthisis, and more than once in patients exhausted by obstinate rheumatism. It may, however, occur in health when the urine is rich in lime and its acidity greatly reduced.

In one or two cases of renal colic the writer has observed numerous shining particles, which upon microscopical examination have been

shown to be an opalescent film covered with small, sharp phosphatic (probably calcium) crystals. (Fig. 243.)

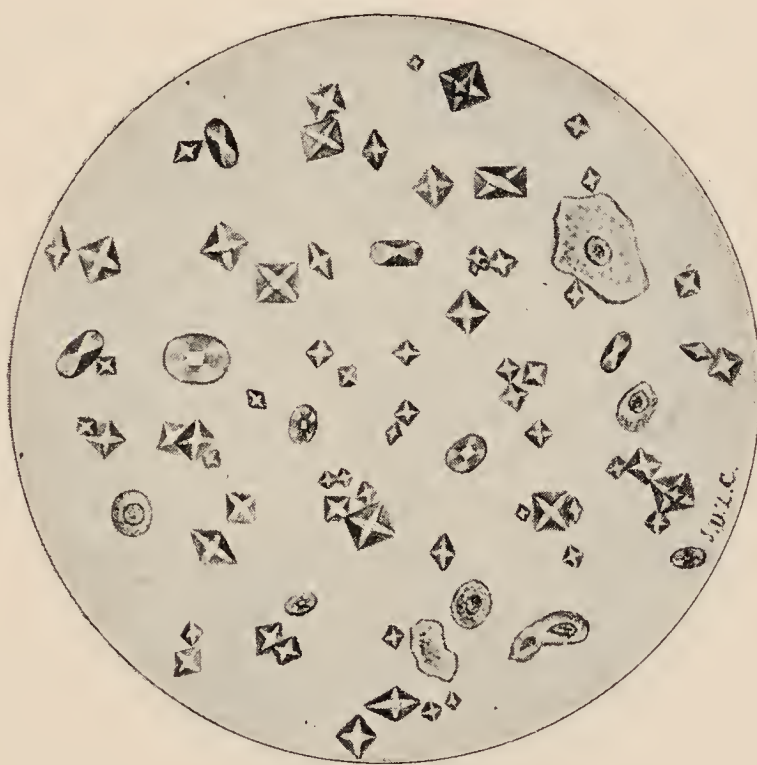
FIG. 243.



Opalescent film in a case of renal colic. (Original.)

Oxalate of Lime. Oxalate of lime occurs in the form of small octahedral crystals, or more rarely as dumb-bells, and in the form of ovals or disks. It is precipitated almost always from acid urines. (Plate XIII.—B, Fig. 2 ; and Fig. 244.)

FIG. 244.



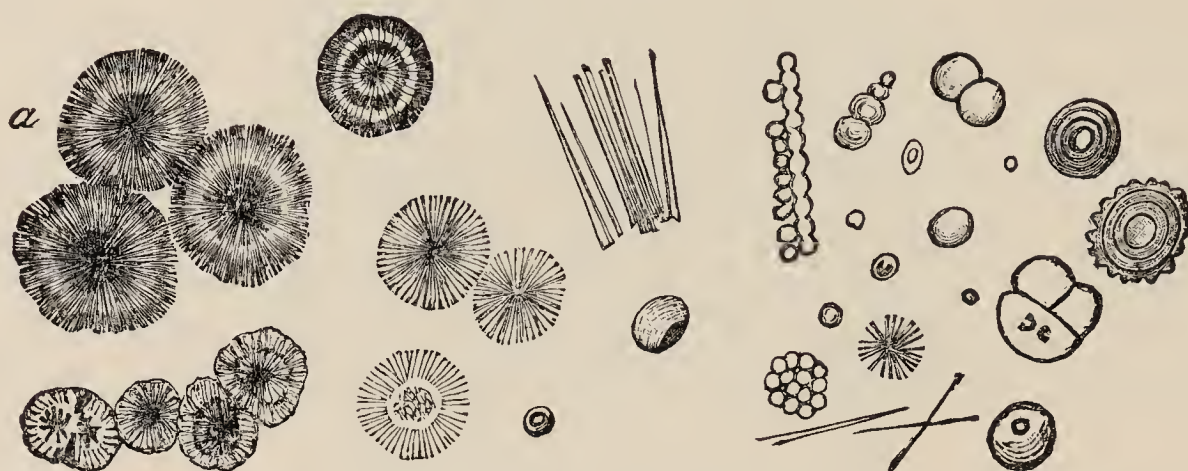
Calcium oxalate.

Oxaluria. According to Beneke, oxaluria has its proximate cause in an impeded metamorphosis, an insufficient activity of that stage which changes oxalic acid into carbonic acid.

When oxalates are constantly found in the urine, a condition of profound hypochondriasis is found to exist, but it has no necessary relation

to the oxaluria. An increase of oxalates in the urine is found in diabetes, especially when there is diminution in the amount of sugar. They are in excess in certain forms of indigestion. Their constant passage may be attended by pains in the back and loins. Flatulent and nervous dyspepsia usually accompany the increase, and neurasthenia also may be present.

FIG. 245.



Crystals of leucin (different forms). (Crystals of creatinin chloride of zinc resemble the leucin crystals depicted at *a*.) The crystals figured toward the right consist of comparatively impure leucin. (From CHARLES: *Chemistry*.)

Cystin. Cystin occurs in the form of hexagonal prisms, either as irregular masses or superimposed upon one another so as to form truncated pyramids. It is a very rare sediment, but appears to be most common in children and young male adults. Several members of the same family have been known to pass it. Its chief clinical significance arises from the fact that rarely it is the basis of calculi.

FIG. 246.



Tyrosin crystals. (Original.)

Leucin and Tyrosin. Leucin and tyrosin are generally described together, though the former is not spontaneously deposited from urine. It appears in the form of spheres, which refract light strongly and have a radiating arrangement. (Fig. 245.)

Tyrosin has been found as a sediment of a light greenish-yellow color in typhoid fever and acute yellow atrophy of the liver. It appears in the form of tolerably long, needle-like crystals, or as bundles and sheaves. Frerichs attaches great importance to leucin and tyrosin in the diagnosis of acute yellow atrophy of the liver. (Fig. 246.)

Cholesterin. This occurs at times in fatty degeneration of the kidneys, jaundice, chyluria, diabetes, and according to Pohl, in the urine of epileptics treated with potassium bromide. (Fig. 247.)

FIG. 247.



Crystals of cholesterin. (Original.)

Melanin. Melanin is held in solution or suspended in small granules. The urine is dark in color, and blackens intensely when sulphuric acid or ferric chloride is added to it. A concentrated solution of the chloride serves to detect the presence of the substance. A few drops added to the urine turn it gray. If a few drops more are added, the phosphates are precipitated along with the coloring-matter. Both are dissolved by an excess of the iron solution. Melanin is usually found in cases of melanotic carcinoma.

CHAPTER XLIV.

THE FECES.

General Considerations and Macroscopical Appearance.

THE number of stools in health varies chiefly with the individual and the character of the food taken. After infancy one passage in twenty-four hours is the rule, but it is natural for some persons to have two or three; and for others to have but one passage in two, three, or four days. Such a condition is termed constipation, while pathological constipation is properly called *obstipation*. The opposite condition is known as diarrhœa. The amount and character of food and drink ingested influence the number of stools. Exercise also plays a role; increased or diminished peristalsis, from whatever cause, will induce diarrhœa or constipation respectively. In disease both extremes are observed—absolute non-passage of feces for days, as in obstruction, or an almost continuous discharge, as in some forms of intestinal inflammation. It is well to remember that diarrhœa may be the symptom of obstipation, as when impacted feces in typhoid causes looseness of the bowels.

Quantity. The quantity of feces varies with the quantity and nature of the food. If most of the food is digested, there will be but little left to form feces. In any disease that prevents the absorption of digested food or causes an increase in the fluid contents of the intestine, as cholera, the amount of feces will be increased. In health about 140 to 200 grammes are voided in twenty-four hours.

Form. The form and consistence of healthy stools vary somewhat. They are commonly cylindrical and firm or mushy. When they remain long in the intestinal canal and the water is extracted, they become hard and may form balls, or flattened masses known as scybala. These are frequently seen in convalescing typhoid patients. On the other hand, the feces may be without form, and are then liquid, either watery as in cholera, or purulent or bloody. Many diseases cause such a condition.

Odor. The odor of feces is sometimes more or less characteristic of certain conditions. Thus the stools of nursing infants have a sour smell, while in infantile diarrhœa and when fermentation takes place, they have an odor of sebacic acid. When urine is mixed with the stools, the odor is ammoniacal; when blood is present, the odor is often stale.

Reaction. The reaction is not constant. In intestinal catarrh with acid fermentation it is acid, and in alkaline fermentation it is alkaline.

Color. The color of the stool varies too much to be of diagnostic value. When much meat is eaten, the color is very dark, chiefly owing to the presence of hæmatin and sulphide of iron. A lighter color is seen when a vegetable diet is taken, the color according to Fleischer, being

then due chiefly to urobilin. With absolute milk-diet the color almost disappears. Unaltered bile-pigments are never found in the feces normally. The ingestion of some varieties of fruit, notably huckleberries, and of certain drugs as iron and bismuth, renders the stools black. Calomel causes green stools, on account of the biliverdin discharged. Green stools may also receive their color from the presence of a bacillus which produces a green pigment. Santonin, rhubarb, and senna cause yellow, and hæmatoxylin red stools. The last fact is important, as parents or nurses should always be warned to expect red passages when hæmatoxylin is given.

The feces may be red or reddish from the presence of unaltered blood; or black when the blood has undergone changes—the so-called “tarry stools” are of this character. With a decrease in the amount of bile the stools become less highly colored; and if the bile is cut off, they become clayey. This color may in some cases be due to the presence of fat left undigested because of the lack of bile. On the other hand, if from disorders of the stomach and intestine the contents pass through too rapidly, the feces may contain unaltered bile and unchanged bile-pigment, thus giving a green or yellow color and showing the bile-reaction.

Constituents. The constituents of feces that can be recognized by the naked eyes are numerous. Seeds, stones, skins of fruit and berries, and the fibres of vegetables are often seen in normal stools. In the passages of children and weak-minded individuals foreign substances of all descriptions may be present. Foreign bodies and partially digested portions of food may be mistaken for parasites. Portions of tumors from the digestive tract may appear in the feces.

In certain diseases of the stomach and small intestine, and in those who eat very fast and do not properly masticate their food, undigested and unchanged particles of food may be seen in the stools.

Shreds of mucous membrane of varying size are passed with the feces. Von Jaksch saw such a shred 5 cm. long and 3 cm. broad in a case of cholelithiasis. Various sized pieces of membrane, consisting of transformed mucus, are passed in membranous enteritis. Particles resembling sago-grains, perhaps the result of over-indulgence in farinaceous food, have been found.

Gallstones in the feces have great clinical value. They may escape detection if not properly sought for. When suspected, each stool should be passed through a linen sieve, the fecal masses being softened with water. The gallstones may be found as small crumbling masses, composed chiefly of cholesterin (intrahepatic calculi), or as hard, irregular, smoothly worn, shining, and many-sided stones, sometimes as large as an egg, usually the size of a pea. Enteroliths are occasionally seen. They are said to originate in the appendix.

Blood may be present in the feces in varying proportions and conditions. When found unaltered on the surface of scybalous masses, it is from the rectum or large intestine, and probably the result of traumatism. Bleeding piles may cause such an appearance, and may even cause very free hemorrhage. Severe hemorrhage may come from ulceration of the rectum or colon due to malignant disease or severe inflammation. The blood

may be intimately mixed with the feces, and have its origin in the large intestine; but much more commonly its source is in the stomach or small intestine. Under such circumstances it is nearly always more or less changed by the intestinal juices, and is brownish red or black (the tarry stool mentioned above), or has the appearance of coffee-grounds. The brighter the color of the blood, the nearer is the source of hemorrhage to the anus. The more retarded the passage the greater the change; while, if quickly expelled, blood from the small intestine may be passed unchanged, as in the hemorrhage of typhoid fever. The microscope may detect blood when the naked eye fails, but the blood-corpuscles are usually destroyed unless the hemorrhage is large and quickly discharged. The hæmin and guaiac tests or the spectroscope may be used. It must be remembered that the hæmin test sometimes fails even when blood is present, and that blood may be found in small amounts as a result of eating raw meats, sausages, and the like. It is also to be remembered that certain drugs, as already stated, may color the feces red, and simulate blood.

Mucus may be present in the stools in health, but when in any marked quantity there is a catarrh of the mucous membrane of the intestines. When hard scybala are covered with mucus or the mucus is seen in shreds, the large intestine is the seat of a catarrh; although mucus may be mixed with thin stools, as in dysentery. Usually, however, when the mucus is finely divided and mixed with the feces, it comes from the small intestine. Mucous shreds have already been mentioned. In cholera the particles of mucus look like boiled rice, hence the term "rice-water stool."

Fatty stools, to the naked eye, appear greasy, or even clayey when there is much fat, even though bile-pigment may be present.

Pus may be present in large quantities from rupture of an abscess into the intestinal tract, or when there are ulcerations from various conditions, producing pus in considerable quantities.

Microscopical Examination of the Feces.

Many animal parasites are not microscopic, but it is convenient to consider them in the following paragraphs. A small portion of the solid feces to be examined is placed on a slide, moistened with a 0.5 per cent. salt solution, and a cover-slip applied; or if liquid, various drops are to be examined. The constituents found will vary with the food taken as well as with disease.

Constituents Derived from Food.

There may be portions of digested or undigested food. In general it may be said that the presence of large pieces of unchanged food, or many small particles of undigested or only partially digested food, indicate defective digestion in the stomach or small intestine. If unchanged bile is present, some particles will be colored yellow—another indication of disordered function.

We may see muscle and elastic fibres in more or less abundance according to the quantity of meat eaten by the patient. The former are

recognized by their transverse striation; the latter, by their double contour and curling ends. Fat may be present as fatty globules or in the form of needles—fatty crystals. Much fatty food increases their number, and they are seen plentifully in alcohol-poisoning, in jaundice, in pancreatic diseases, tuberculosis of intestines, diseases of the mesenteric glands, and enteritis. The crystals may be transformed into fat-drops by the addition of acid and heat. When meat is eaten freely, areolar tissue may be present, but its presence otherwise points to defective digestion. Schmidt considers that if remnants of areolar tissue are present after a test-meal of 100 grammes of chopped meat, it points to defective gastric digestion, while the presence of undigested muscular fibre indicates disturbance of intestinal digestion. Various forms of vegetable cells are commonly seen, in which granules of starch may be contained or the starch particle may be free. The presence of starch-granules or of many vegetable remnants containing starch, or a general blue color on the addition of iodine, always indicates imperfect starch digestion. Normally, starches are practically completely digested. Undigested milk occurs in the stools of children and when diarrhoea prevails; a substance, probably casein, has been described by Nothnagel as occurring in the feces of persons who have intestinal disturbances.

In persons living on vegetables most of the above constituents will be absent, and in infants who partake only of milk, the derivatives of meat are absent, while there will be an excess of fatty crystals, fat-globules, and coagulated products.

Constituents from the Alimentary Tract.

Epithelium. In every normal stool will be found *epithelium* of the squamous variety. Occasionally the columnar form is seen, and modified epithelial cells are very common. In intestinal catarrh their number is greatly increased.

Red Blood-corpuscles. In the majority of blood-stained stools red blood-cells are not found; in their stead will be seen masses of free blood-coloring matter and rhombic crystals of hæmatoidin. Red cells are seen in dysentery, in bloody stools in which the blood comes from near the anus, as in hemorrhoids, and when blood is discharged with the feces soon after the occurrence of the bleeding. If there is any doubt as to the presence of blood, when the corpuscle can not be found, a true decision can sometimes be reached by examining for hæmin crystals by Teichmann's method. A portion of feces is dried and powdered, placed on a slide with a grain of common salt, and covered by a cover-slip. A few drops of glacial acetic acid are directed beneath the slip, the slide is heated just to boiling, and if blood has been present, reddish-brown rhombic crystals of hæmin will usually soon be found; but, as stated, the test is not wholly reliable.

Leucocytes. Leucocytes are frequently seen in normal stools; when pus is present or discharged into the intestinal canal, they are found in great numbers, as in ulceration of the intestine and in abscess.

Molecular débris, or detritus, occurs in all feces as part of the waste-products.

Crystals. *Fat crystals* are the most important. They have been quite fully considered above. There seems to be little doubt that the crystalline needles found in the feces are salts and fatty acids, and not tyrosin.

FIG. 248.



Collective view of the feces. (Eye-piece III., objective 8 A, Reichert.) *a.* Muscular-fibres. *b.* Connective tissue. *c.* Epithelium. *d.* White blood-corpuscles. *e.* Spiral cells. *f, i.* Various vegetable cells. *k.* Triple phosphate crystals in a mass of various micro-organisms. *l.* Diatoms. (VON JAKSCH.)

Charcot-Leyden crystals, similar to those already described (see Sputum), have occasionally been met with in the stools of typhoid fever patients, in dysentery, intestinal tuberculosis, and ankylostomiasis.

Hæmatoidin crystals occur as reddish-brown, hard, needle-shaped bodies, usually in clusters, and free or enclosed in masses of mucin or a substance resembling it. They have been found in the feces of breast-fed infants, in cases of chronic intestinal catarrh, and, by von Jaksch, in the stools of a case of nephritis.

Crystals of various *salts of calcium*, of *triple phosphate* and *cholesterin* will often be recognized, but they have no diagnostic value. When bismuth is being administered, black rhombic crystals of the sulphide of that metal will be recognized.

Parasites.

Animal and vegetable parasites flourish in the intestinal tract, and the presence of some of these in the feces is of the greatest clinical importance.

A. Animal Parasites. Following Leuckart's classification, we will consider these parasites under the secondary heads:

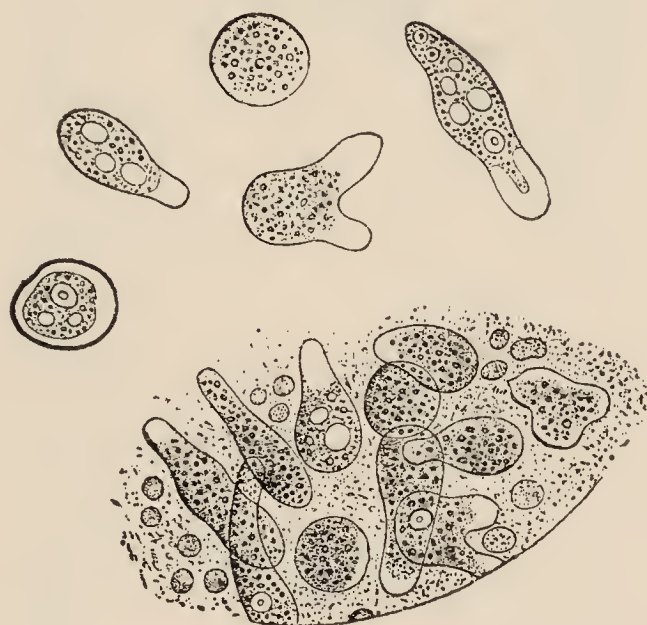
I. Protozoa. 1. RHIZOPODA. This variety is made important because *Amœba dysenteriae* or *Amœba coli* belongs to it.

a. Amœba Dysenteriae—Amœba Coli. This protozoön has been found so many times by various observers in different parts of the world that it is now considered by many writers to be the causative factor of so-called tropical dysentery. The subject has received special study in this

country by Osler,¹ Stengel,² Dock,³ and Councilman and Lafleur.⁴ The work of Councilman and Lafleur is at the present time the best that has been published in any country; and to it the reader is particularly referred. The following notes are based on this book:

Amœbæ dysentericæ vary in size from 0.012 to 0.035 mm. They are found most plentifully in the small gelatinous masses often to be seen in the feces. They vary in number in different cases, and in the same case at different times. The severer the lesions the more numerous are the amœbæ. When not active, they are round or oblong, and highly refractive. They contain one or more vacuoles of varying size. Occasionally the division into an ectosarc and endosarc is easily made out. When thus inactive, they may be confounded with swollen connective-tissue cells and compound granular bodies found in feces. The active amœbæ have, however, a characteristic movement. This consists of progression and of thrusting out and retraction of pseudopodia. Their activity varies greatly. It is best seen when the body heat is maintained. The stools should be passed into a clean and warm pan, and examined immediately or kept

FIG. 249.

*Amœba coli.* (HALLOPEAU.)

warm until examined, and a warm stage should be used with the microscope. The division into ectosarc and endosarc is usually clear during activity. The ectosarc is composed of a hyaline homogeneous mass, as are the pseudopodia, while the endosarc is made up, not of granular matter, but of a dense homogeneous mass enclosing vacuoles and a nucleus. The vacuoles may vary in size as well as in number. There may be one or two large ones, or the entire endosarc may appear as made up entirely of small vacuoles. The nucleus is sometimes plainly seen as a small rounded body, but is more often difficult to distinguish from the vacuoles. Dried cover-slip preparations may be stained with the various aniline dyes, but the results are not satisfactory.

The amœbæ will often be found to enclose bodies such as red blood-corpuscles, pus-cells, blood-coloring matter, bacilli, and micrococci.

¹ Johns Hopkins Hospital Bulletin, May, 1890, vol. i., No. 5.

² Philadelphia Medical News, 1890.

³ Texas Medical Journal, April, 1891.

⁴ Johns Hopkins Hospital Reports, vol. ii., Nos. 7, 8, 9.

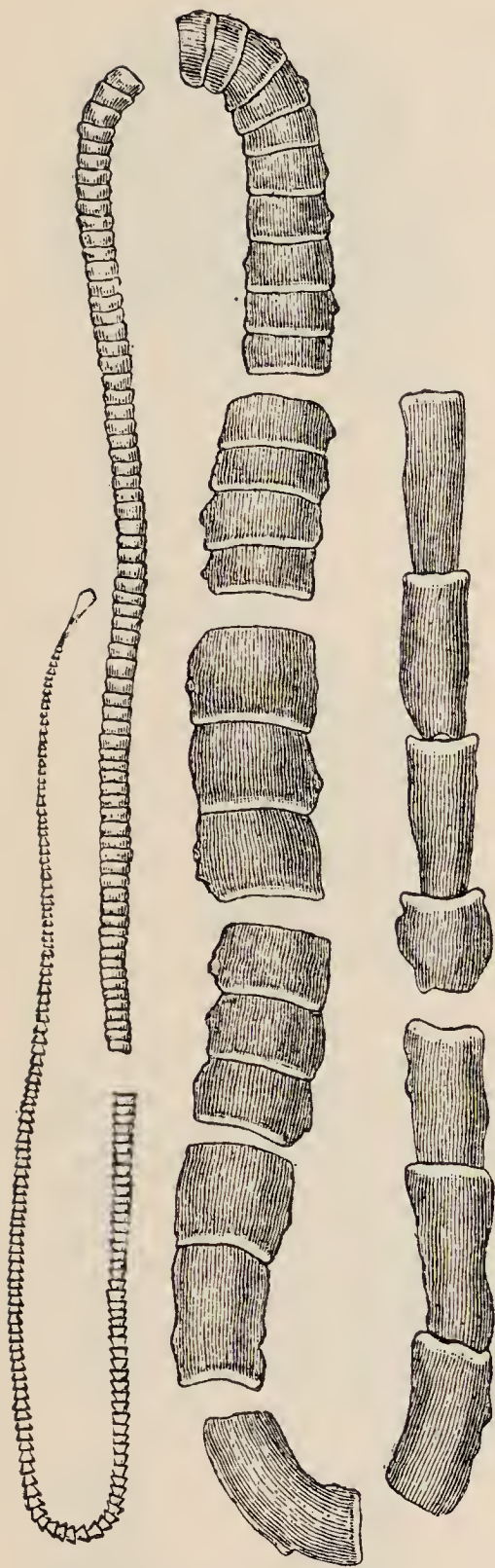
In examining feces for *Amœbæ dysentericæ* the suggestion given above concerning the warm bed-pan and warm stage of the microscope, and, above all, the immediate examination of the stool, should be adhered to. The small gelatinous masses should be selected when present. In obtaining the material it is often of advantage to pass well into the lower intestine an ordinary rectal tube, in the eye of which more or less of the mucous exudates from the surface of the gut is likely to adhere, suitable particles from which may be employed for the microscopical examination. In some cases of chronic amœbic dysentery, especially when much thickening of the wall of the colon exists, one may fail repeatedly to find the parasites in the ordinary dejection; but may succeed excellently if a saline aperient be given and the watery dejecta following this be used for examination. In searching for the amœbæ it is usually sufficient to make use of the ordinary laboratory powers of the microscope; but for the details of structure it is advisable to have recourse to the $\frac{1}{12}$ inch oil immersion lens. It is desirable to encounter the amœbæ in active movement, in order to appreciate the number and character of the pseudopods, and the degree of mobility of the parasite, these features being utilized in the positive recognition and classification of these organisms. It should be advised therefore that at regular intervals, of half minute intermission, hasty sketches be made of any amœboid body found, in order that the best and a fixed appreciation of the above characters may be had.

2. SPOROZOA: *Coccidium perforans* (Leuckart) (*C. hominis*) has been met in the stools of human beings occasionally. These sporozoa, which in their earlier stages have existed in the lining epithelial cells of the intestine, at the stage of encystment escape into the intestinal lumen; and may be found in large numbers in the dejecta. They are short elliptical bodies, very like the familiar *Coccidium oviforme* of the liver of rabbits, but smaller in size.

3. INFUSORIA. *a. Cercomonas intestinalis* (vel *Lambliæ intestinalis*, etc.) is a pyriform protozoön, about 10 or 15 μ in length, nucleated, with a marked mouth-like depression on one side anteriorly, and provided with four pairs of flagella, three pairs symmetrically placed about the border of the depression mentioned and the fourth pair containing the posterior pointed extremity. While more frequent in the lower animals, it has often been encountered in man. Its most frequent habitat is in the duodenum and jejunum, but occurs also in the large intestine. In the stools it is often in the encysted stage, but may be encountered in an active state. It commonly occurs in the stools of chronic diarrhœa, but is of uncertain pathogenic importance.

b. Trichomonas intestinalis (vel *Tr. hominis*, *Cercomonas intestinalis*, *C. hominis*, etc.) is somewhat smaller than the preceding, being about 10 or 12 μ in length, of a pyriform or subovoid shape, tapering at each extremity, and provided with five flagella (four attached to the anterior extremity and the fifth passing from the anterior part backward as the border of an undulating membrane and extending some distance beyond the posterior extremity of the cell). The appearance of cilia is given along the side by the undulating membrane. Close to the base of the flagella is a mouth-like depression. Within the cell a definite nucleus

FIG. 250.

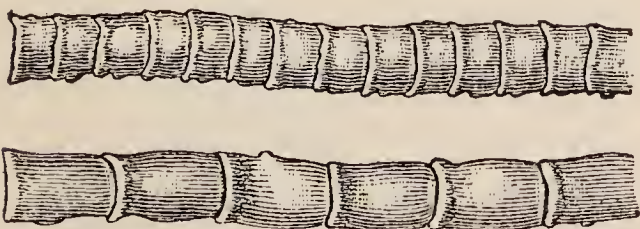


Tænia saginata: natural size.
(After LEUCKART.)

FIG. 251.

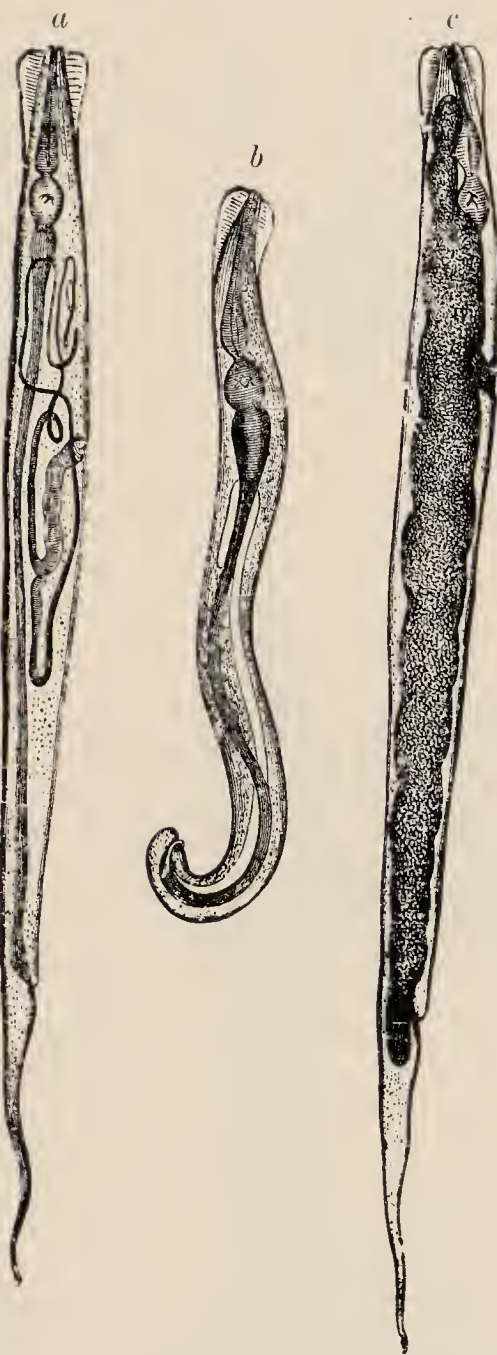


Ova of *T. solium*: *a*, with yolk; *b*, without yolk, as in mature segments. The hard brown shell is indicated. (LEUCKART.)



Half-grown and mature proglottides of *Tænia solium*: natural size. (After LEUCKART.)

FIG. 252.



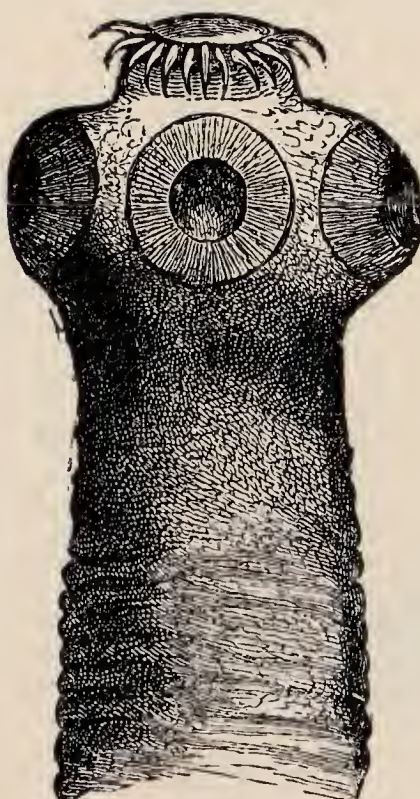
Oxyuris vermicularis, magnified: *a*, young female; *b*, male; *c*, mature female, full of eggs. (PAYNE.)

FIG. 253.



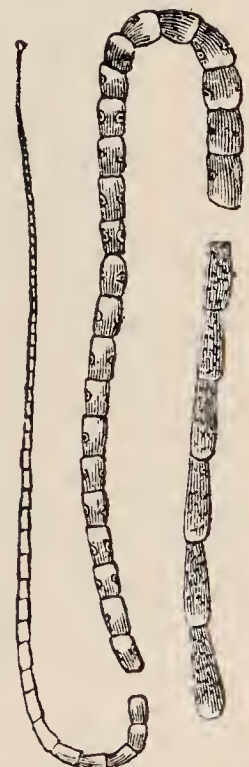
Uncinaria duodenalis, magnified: *a*, female; *b*, male. (BRISTOWE.)

FIG. 254.



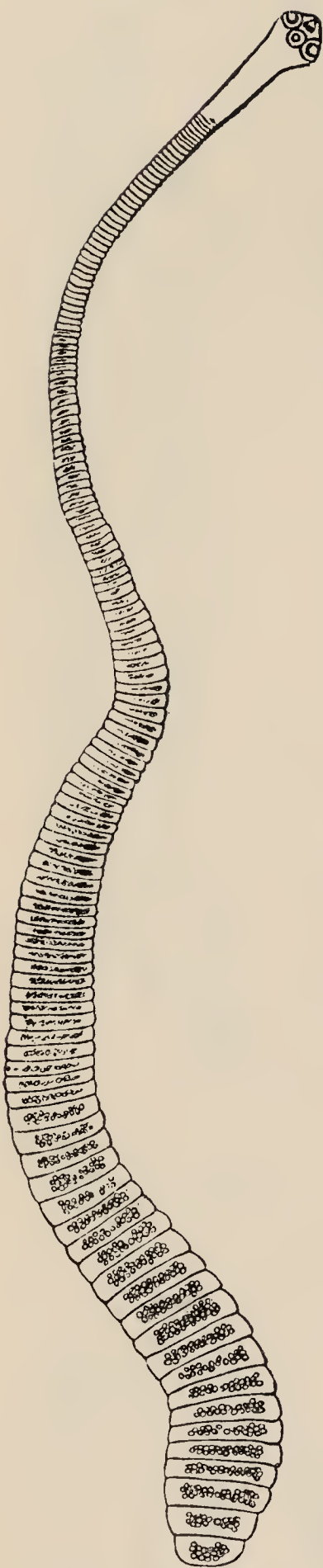
Head of *T. solium*: $\times 45$.
(LEUCKART.)

FIG. 255.



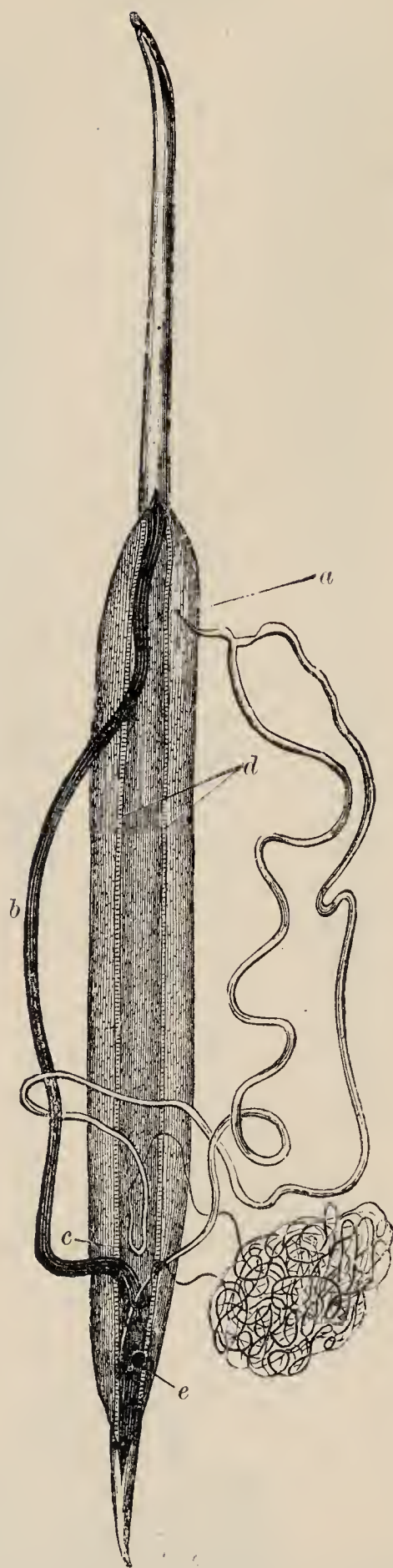
Tænia cucumerina: natural size. (After LEUCKART.)

FIG. 256.



Hymenolepsis nana : $\times 12$.
(After LEUCKART.)

FIG. 257.



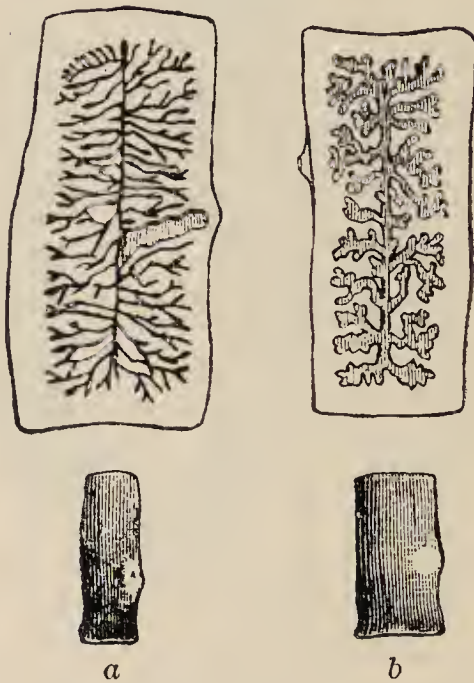
Ascaris lumbricoides, dissected and
walls thrown back : a, genital orifice;
b, intestine ; c, oviducts ; d, longitu-
dinal band ; e, ovaries. (HELLER.)

FIG. 258.



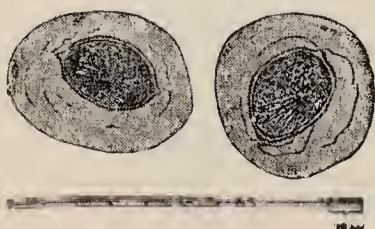
Head of *Tænia saginata*.
(EICHHORST.)

FIG. 259.



Proglottides of (a) *Tænia sagi-
nata* and (b) *T. solium* : natural
size, and enlarged three times
to show arrangement of uterus.
(After LEUCKART.)

FIG. 256a.



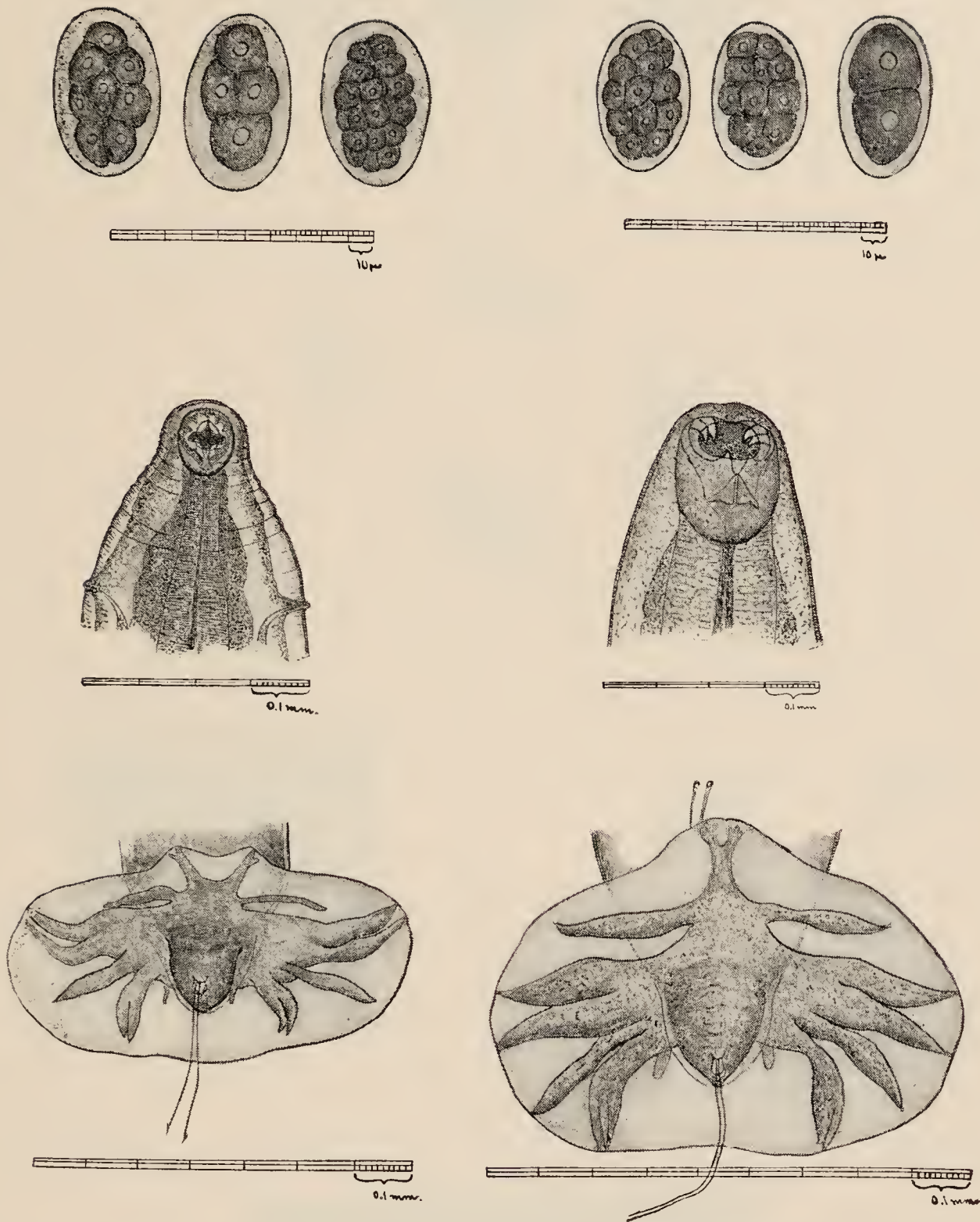
Ova of *Hymenolepsis nana*. (Smith.)

FIG. 260.



Proglottis of *Bothriocephalus
latus* : natural size, and enlarged
three times. (After EICHHORST.)

FIG. 261.

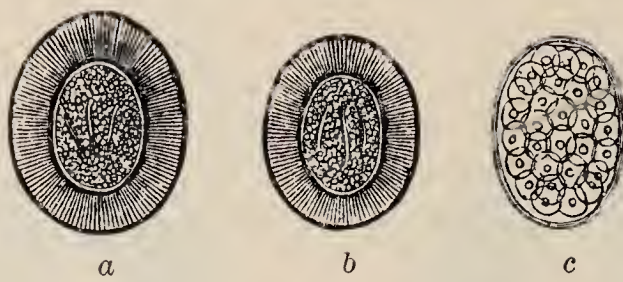


Above: to the left, three ova of *Uncinaria Americana* in different stages of segmentation, from $66\ \mu$ to $68\ \mu$ in long diameter: to the right, three ova of *Uncinaria duodenalis* in varying stages of segmentation, long diameter from $58\ \mu$ to $62\ \mu$.

Middle: to the left, the head of *Uncinaria Americana* (dorsal view), exhibiting the smaller size of the head and more tapering anterior extremity of the worm in comparison with *U. duodenalis*, and its buccal armature consisting of lips; to the right, the head of *Uncinaria duodenalis* (dorsal view), drawn to same scale, showing its greater size and the hook armature of the buccal border.

Below: to the left the caudal border of male *Uncinaria Americana* (dorsal view, dorsal lobe of bursa thrown forward), showing comparative size of this worm in relation to that of *U. duodenalis*, partly divided dorsal lobe and suggestion of a ventral lobe, the dorsal ray divided nearly to base, each of its divisions having a bipartite tip, showing also the tip of tail within bursa, the long barbed sexual spicules, and the arrangement of the rays of the bursa; to the right, caudal bursa of male *U. duodenalis*, showing larger size of bursa, entire dorsal lobe, absence of ventral lobe, division of dorsal ray limited to distal third and tripartite tips of each division, with other features in general similar to *U. Americana*. (SMITH.)

FIG. 262.



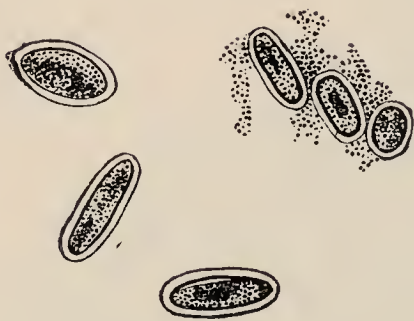
Eggs of (a) *T. saginata*; (b) *T. solium*; (c) *Bothriocephalus latus*: $\times 300$. (After EICHHORST.)

FIG. 263.



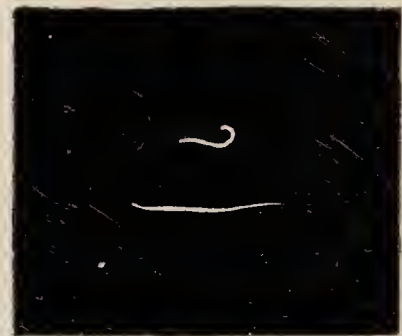
Eggs of *Trichuris trichiura*: $\times 275$. (EICHHORST.)

FIG. 264.



Eggs of *Oxyuris vermicularis*: $\times 275$. (After EICHHORST.)

FIG. 265.



Oxyuris vermicularis: natural size. (EICHHORST.)

FIG. 266.



Trichuris trichiura, male and female: natural size. (EICHHORST.)

FIG. 267.



Uncinaria duodenalis, with eggs. (After BLICKHAHN.)

with nucleolus may be distinguished. It is usually encountered in cases of chronic diarrhoea, but its pathogenic influence is doubtful.

Balantidium coli (vel *Paramœcium coli*) is a large ciliate infusorian, of a somewhat flattened oval shape, measuring from 70 to 100 μ in its long diameter. It is colorless and uniformly covered with cilia, having a special border of cilia at the anterior (blunt) extremity. Its interior is finely granular, contains two contractile vacuoles, a bean-shaped nucleus, and a variable amount of extraneous substances which have been ingested from the intestinal contents. In general it closely resembles the ordinary "slipper animalcule" of the gutter. It is found, like the preceding infusoria, in the stools of patients with chronic diarrhoea, but its influence in the production of the condition is doubtful.

II. Vermes. Verminous intestinal parasites are more generally known than the above protozoa, and their clinical importance is less uncertain. Both local and general symptoms of importance may be predicated of individual examples. They are most conveniently outlined following the usual classification of flat and round worms:

1. PLATODA. 1. *Tapeworms—Cestoda.* These parasites infest only the small intestine, to the walls of which they cling by the head. The head and neck are small; the joints are flat and form long ribbons. The distal joints continually drop off and can easily be recognized in the stools by the naked eye, and the eggs by the use of the microscope. The feces are best washed in water and broken up to obtain the eggs. As the lower joints are lost, new ones take their place from above. The more important are the following:

a. Taenia solium reaches a length of 2 to 3 metres. The head is the size of a pin-head. The neck is 2.5 cm. long, as thick as a thread, and without joints. The segments forming the body are short and broad near the neck, but as they increase in size there is more growth in length than in width. The average dimensions are 9 to 10 mm. by 6 to 7 mm. The head appears dark, the body white. The joints are easily detected in the feces by the naked eye. Under the microscope the head is seen to be spheroidal, with 4 pigmented sucking-disks surrounding at the base a rostellum, which is a "crown of hooks"—chitin hooks—about 24 in number. In the ripe segments, or proglottides, is seen the longitudinal uterus with about 12 horizontal ramifications to a segment. The eggs are round or oval, 0.035 mm. long, with a thick striated shell when ripe, and contain hooklets.

b. Taenia mediocanellata or *saginata*. This worm is 4 or 5 metres long. The head is slightly larger than that of *T. solium*, and more pigmented, and the segments are longer, thicker, and darker. The head is supplied with 4 powerful sucking-cups, but has no rostellum or hooklets. The uterus in the ripe segment is much more finely branched than in the *solium*, and these segments have independent movement. The eggs are very similar to those of *T. solium*, but may be somewhat larger.

c. Taenia Echinococcus. This tapeworm in its adult stage is a common parasite of the intestine of dogs. In its embryonic stage the bladder worms are ordinarily found in the viscera or flesh of man, and a number of the mammalia. The cysticercus is peculiar in that it is capable of

multiplication to the formation of large collections of the bladder worms, thus constituting the so-called "hydatid cyst." Instances of hydatid formation in the gall-bladder, gall-ducts, appendix vermiformis, or in immediate proximity to the intestine, have been known to present in the feces from time to time the characteristic bladder worms. The cysticercus is a small round or ovoid body, of a somewhat nacreous tint, varying in size from that of a buckshot to that of an ordinary Malaga grape (and even much larger), filled with a clear fluid (or in case of death of the embryo with a somewhat turbid fluid in which the hooklets of the rostellum are to be found) and containing the developed pyriform head of the future tapeworm, with its 4 suckers and rostellum of about 40 hooklets.

d. Hymenolepis nana (vel Taenia nana). In length, *T. nana* is only 10 to 15 mm., and 0.5 mm. in breadth. The round head is but 0.3 mm. in diameter. The segments are all short, and at the lower end of the body are 4 times as wide as they are long. The head is found to have 4 round suckers at the base of a rostellum that can be inverted. At the base of the rostellum are about 22 hooklets. The uterus is oblong and filled with eggs. The eggs have a double membrane. This tapeworm has recently been found in a number of instances in this country. It is especially likely to be found in children. Its ova are nearly spherical, about 40 μ in diameter, colorless, with double wall. The embryo within measures about 22 or 23 μ , is colorless and granular, and is provided with six embryonic hooklets easily discernible. At each pole of the embryo is to be seen a slight protuberance, and between the inner and outer shell membranes, several filaments may be detected. (Fig. 256.)

e. Hymenolepis flavopunctata (vel Taenia flavopunctata). This small tapeworm has been encountered several times in the United States. It attains a length of about one foot. The head is small, clubbed, and unarmed. The neck is short, the developed links 1 mm. in length and 2 to 2.6 mm. wide. The last links are slightly longer and from shrivelling become nearly as narrow as long. Each fully developed link is marked by a small yellow spot in the posterior portion. The ova are similar in appearance to those of *H. nana*, but larger, measuring 60 μ in diameter.

f. Bothriocephalus latus. This is the largest of the tapeworms likely to be met in the human intestine, measuring 7 or 8 metres or more in length. The head is of an elongated club shape, provided with two long, narrow slits, one on each side, serving as suckers. There are neither hooklets nor rostellum. The proglottides are short near the head and very wide, but toward the lower end of the worm become nearly square. Each ripe link shows a reddish or brownish rosette-like mass near the middle, caused by the coiled uterine tube filled with the yellowish or faintly brownish eggs. These eggs may be encountered in the dejecta, as oval, thin-shelled, brownish objects measuring about 70 μ in length and 40 to 45 μ in breadth, showing more or less advanced segmentation and provided with an operculum or lid at one end for emergence of the ciliated embryo. The embryo makes its habitat in the tissues of certain fish.

It will not be necessary to describe certain other varieties that are rarely met with.

Flukeworms, Trematoda. *a. Distoma hepaticum* measures 28 mm. by 10 mm., and is shaped like a leaf. A short head is situated at the broad end and has one sucker; on the under surface is another sucker, and between the two is the opening of the uterus, a highly convoluted arrangement. This worm is not often seen in man. Its habitat is in the hepatic ducts, where it gives rise to the condition known as liver rot. The worm is hermaphroditic; and its eggs after discharge from the genital pore, find their way along the bile-ducts, and into the intestine. They are thin-walled, of a brownish-yellow hue, ovoid in shape, usually seen in advanced segmentation, and measure from 120 to 140 μ in length and 70 to 90 μ in width. A lid may be demonstrated on one end for the emergence of the embryo. The intermediate stages of the parasite are passed in the tissues of snails, and subsequently attached to a blade of grass.

b. Distoma lanceolatum. This fluke is much smaller than the preceding, being more elongated in shape, but preserving the general leaf-shape of the *Distoma hepaticum*. It is about 8 mm. long and 1.5 to 2.5 mm. broad. The extremities, especially the anterior, are sharper than those of the preceding. It is semitransparent, and flecked with brown from the eggs within the body. It is also more common in the lower animals than in man. Its habitat is in the bile passages, its eggs being carried thence into the intestine. They are ovoid, with thin shell, of a light-brown color, measuring about 40 to 45 μ in length and 20 to 30 μ in width. The tiny embryo is ordinarily visible within the granular contents. An operculum may be shown on one end.

c. Distoma sinense (vel D. japonicum). A fluke of about the same size as the preceding, but more pointed anteriorly and more rounded posteriorly than the latter, reddish in color, and when living, nearly transparent. This parasite is found with some frequency—China and Japan—in the human biliary passages. It has also been met in the cat, in the same regions. The eggs may be found in the dejecta, dark (almost black) ovoid bodies, about 30 μ long and 16 or 17 μ broad; they are apt to show in the interior the ciliated embryo. The intermediate life history is not known.

d. Distoma buski (D. crassum). This is a rare human parasite found in the human intestine in China and the neighboring continental and insular districts. A case in an East Indian Lascar sailor dead from typhoid fever, has recently been encountered by Tenille, of Galveston, Texas (personal communication). The parasite is quite large, 3 to 7 cm. in length and 10 to 20 mm. broad. The eggs are 125 μ long and 75 μ broad.

II. NEMATODA. Round worms with complete digestive canal. *a. Ascaris lumbricoides.* This is the parasite usually referred to by the term round worm. It has a light cream color and resembles the common earth-worm in shape. The male worm is about 250 mm. long, and the female 400 mm. The head is made up of 3 prominent lips, and is supplied with microscopical teeth. The vulva is in the posterior third of the body. The eggs are rounded, brownish, 0.06 mm. in diameter, and covered, when fresh, by a rough albuminous coat over a hard shell. This worm has the small intestine for its habitat. It may pass with the stools or work its

way into the stomach and be vomited (the writer has seen them thus vomited during the etherization of a child of ten years). They have been the cause of jaundice by crawling into the ductus choledochus, and may infest the larger hepatic ducts. An enormous number may be present in the intestine at one time.

b. Oxyuris vermicularis. The thread-worm, or seat-worm, inhabits the large intestine, and is often present in the stool as a white, thread-like body; the male 5 mm. and the female 10 mm. long. They often wander out of the anus and into the vagina. The head has a number of small lips, and is covered with a thick skin. The female has 1 vagina and 2 uteri. The eggs are colorless and unsymmetrical, have a laminated shell and a diameter of about 4 mm. They are not often seen in the stools because they hatch out in the intestine.

c. Uncinaria duodenalis (vel Anchylostoma duodenale). This parasite, the European form of hook-worm, is in its parasite life met in the upper part of the human small intestine, where it attaches itself by its special mouth armature to the mucous membrane, wounding the latter and sucking blood from the tissues. Such tiny wounds continue to bleed when the worm changes its places of attachment; and, should the openings be very numerous, through serious and long-continued blood loss, there is apt to be serious, and even fatal anæmia produced. The worm is bisexual, the female measuring from 10 to 18 mm. in length, the male 8 to 10 mm.; both are of a cream-like color, ranging to a faint red (if there be much blood débris in the intestine, individual worms may be blotched with brownish dots and patches). In each sex the head is turned back dorsally, forming the tiny hook from which the common name, "hook-worm," is derived. The head has a cup-shaped mouth, with oval, and at the depth, with pharyngeal armature. The oval armature is of importance in recognition of the species, consisting of a pair of hooklets curving into the mouth from the buccal border on either side of the ventro-median line, and a dorsal pair of prominent tooth-like structures at the dorso-median line—the appearance being given of six hooklets at the border of the buccal capsule. The female ends posteriorly in a finely tapering manner, the male in a blunt point surrounded by a lobed bursa supported by characteristic rays. The vaginal opening is at about the posterior third of the length of the worm. The ova are colorless, oval in shape, thin walled, with more or less segmented contents, and measure 50μ in length and 32μ in width. These ova develop after discharge upon some moist spot into minute rhabditiform embryos, capable of independent existence in dirty water or moist soil; in their free life they undergo several moults. They are known to be capable of gaining entrance to the intestine of the future host by penetrating through the skin (Loos), and in unknown manner making their way to the proper intestinal habitat. In so doing it is believed they are responsible for certain itch-like lesions, known as ground-itch or water-itch. It is possible that they also gain entrance to the intestine of the host by the direct alimentary route as in swallowing the dirty water in which these embryos live.

d. Uncinaria Americana. Recently in the West Indies and our

Southern States a parasite almost identical with the above, has been found, and has by Stiles been classified as species distinct from the European hook-worm. Its effects are similar to those of the latter. Its habitat is similarly in the upper portion of the human small intestine. It is slightly shorter than the above measurements given for the European hook-worm and is distinguished from the latter by the fact that instead of possessing the hooked buccal armature of the head, the border of the capsule on each side of the ventro-median line projects into the mouth as a large uniform semilunar lip, a smaller lip of similar appearance existing on either side of the dorso-median line. Other points of difference may be noted in the rays of the caudal bursa of the wall, and in the fact that the vaginal opening of the female is nearer the middle of the worm than in the European species. The ova are somewhat larger characteristically, but otherwise have the same appearance. They measure 60 to 68 μ in length by about 40 μ in width. The intermediate stages and mode of infestation of the host have not been completely discovered, but probably are similar to those of the European form.

e. *Trichinis trichinora* (*Trichocephalus dispar*). The common whip-worm, so-called from its shape is 4 to 5 cm. in length, the female being longer than the male. It is recognized by the marked contrast between the anterior and the posterior portions. The former is thin and thread-like, the latter expanded and broad, and in the male curled up. The eggs are brownish, about 0.05 mm. long and half as broad, and have a button-like projection at either end; they are to be recognized in the stools, where large ones may be present. There may be only a few or thousands of the forms present in the body. They live chiefly in the cæcum and large intestine. They have been thought to cause beri-beri by some writers.

f. *Trichina spiralis*. It is the adult trichinæ which exist in the intestine and are found very frequently in the feces. These produce the embryos, which become muscle trichinæ. The adult male is 1.5 mm. long and the female twice that length. The former has 2 projections from the posterior extremity, between which are 4 papillæ. The female has a tubular uterus and a tubular ovary in the posterior half of the body.

g. *Strongyloides intestinalis* (vel *Rhabdonema intestinale*). The parasitic and the free generations of this worm in the older texts have been regarded as separate species, the parasitic form being known as the *Anguillula intestinalis*, the free form (found in feces after development outside the body, or in mud) as *Anguillula stercoralis*. The parasitic form is a parthenogenetic female, a little over 2 mm. in length, cylindrical, slender and finely tapering posteriorly. The vulvar opening is at the posterior third of the body. A large number of these may infest the intestine. Ova of a greenish-yellow color, oval, and of advanced segmentation, are deposited, which quickly (several hours at most) hatch into embryos of a rhabdite type. These at time of birth are from 200 to 240 μ long, and about 12 μ broad; when found in fresh stools they are further grown, 500 to 600 μ long and 16 to 20 μ broad. Under favorable conditions these develop into sexually mature rhabditoid males and females (*Anguillula stercoralis*), and the females deposit ova or living

young, very like those produced in the intestine but a little more slender, which if circumstances permit gain access to the digestive tract of another host where they pass their parasitic life as *Anguillula intestinalis*. Their pathogenic influence is uncertain.

D. *Rhabdonema*. *Strongylides*. Under *Rhabdonema intestinale* we now include two small nematodes, which were termed *Anguillula intestinalis* and *A. stercoralis*, and which are probably one and the same. They are found in the stools of cases of endemic diarrhoea of hot countries. Usually the young embryos, which have developed in the intestinal canal, are dejected with the stools. These sexually mature embryos are 0.8 and 1.2 mm. long, male and female respectively. They are round and have a cone-shaped head. There are 2 jaws and 2 teeth in each. The adult worm is about 2.2 mm. long and 0.04 mm. thick. The mouth has 3 lips. The vulva is at the beginning of the posterior third. The eggs might be easily confounded with those of *Ankylostomum duodenale*, but are somewhat more pointed, and larger. The rhabdonema infests the small intestine, and is frequently found in connection with ankylostoma. Echinococcus hooklets and portions of the striated cyst-wall have been found in the feces. The rupture of a hydatid cyst into the intestine may be discovered when the above structures are found, pointing to a cyst in the abdominal cavity.

B. Vegetable Parasites. We find both *pathogenic* and *non-pathogenic* vegetable parasites in the feces. The latter we have classed as (1) moulds, (2) yeasts, and (3) fission-fungi.

1. **Moulds.** The only mould found in the stools is the thrush fungus when children are the subjects of thrush in the mouth. It is of rare occurrence in the feces, and has no special clinical import.

2. **Yeasts.** In all feces, in health or disease, yeast-fungi exist. They are most numerous in acid stools. They are round or ovoid, and usually occur in groups. They stain dark brown with a solution of iodine and potassium iodide, while apparently similar cells become violet or blue with the same dye.

3. **Fission-fungi.** Bacteria are found in greatest numbers in the feces, chiefly as bacilli, micrococci, and spirilla. They may be grouped as torulæ or sarcinæ. They present active movement, and may be separate or in colonies. *Bacillus coli communis* (*B. termo*) is the most frequent form met with both in health and disease. It is not yet determined what relations it holds to normal and abnormal conditions, or what is the true relationship between it and certain other bacteria. *B. subtilis* is another bacterium found both in health and disease. As above stated, there are various organisms which stain brown with iodo-potassic iodide solution, and others which become blue with the same dye. Von Jaksch has studied these latter closely. They assume various forms, as long or short rods, and stain different shades of blue or violet. One of them is *Clostridium butyricum* of Nothnagel. It occurs as large round cells, like yeast-fungi, and stains like the tubercle bacilli with the Ziehl-Neelsen fluid. Von Jaksch finds these fungi in greater abundance in intestinal catarrh. They are present in both acid and alkaline stools.

Bacillus coli communis has been found in the blood, in various organs,

in feces of cholera patients, in healthy feces, in the air, and in putrefying infusions ; it can also be found in the peritoneal exudate in most cases of peritonitis.

Morphology. A bacillus, $4\ \mu$ to $6\ \mu$ by $2\ \mu$ to $3\ \mu$, with rounded ends, in cultures sometimes a short oval. Five or more flagella have been observed attached to the organism.

The remaining fungi on the list are pathogenic.

Spirillum Cholerae Asiaticæ. (See page 797.)

Spirillum of Cholera Nostras. *Morphology.* Longer and thicker than the spirillum of Asiatic cholera ; central part thicker than ends. Stains like the true cholera spirillum.

Biological Properties. Culture. A thick, stocking-like funnel of liquefaction instead of a fine, straight funnel.

Typhoid Fever Bacillus. This bacillus is present in the stools of typhoid fever patients, but can not be directly differentiated by microscopical examination alone, either when stained or unstained. It is necessary for its detection to make pure cultures according to bacteriological methods. The bacillus is about as long as the tubercle bacillus, but much thicker, being one-third as thick as it is long. The ends are rounded. It is best stained by concentrated aqueous solutions of methylene-blue, the dried preparations on the cover-slip being prepared as above. (See Typhoid Fever.)

Tubercle Bacillus. The bacillus of tuberculosis is frequently found in the feces of persons suffering from intestinal tuberculosis and occasionally in the feces of cases of pulmonary tuberculosis, when sputum has been swallowed. When tubercle bacilli are constantly found in the feces, and in large quantities, it points to the former condition almost to a certainty. They are detected by the methods employed in the examination of sputum.

Bacillus of Shiga in Tropical Dysentery and Summer Diarrhœa. The organisms have been found by Shiga and Flexner in children. Some of them resemble very closely *Bacillus coli communis*. Bacillus A is a bacillus with rounded ends, $3\ \mu$ to $4\ \mu$ by $0.7\ \mu$.

Chemical Examination of the Feces.

The chemical examination of the feces has but slight clinical value, with the exception of the rather elaborate procedures necessary in quantitative fat estimations. Mucin and albumin are normally present ; peptones in different diseases. (Von Jaksch.) Among the acids to be found are bile-acids, volatile and fatty acids, formic, acetic, butyric, and propionic acids ; while phenol, indol, skatol, cholesterin, and fats, according to the same author, are always present. The demonstration of these substances will not aid in the diagnosis.

The normal *coloring-matter* of the feces is urobilin and pigments derived from the food. The presence of urobilin may be shown by the proper tests. As before stated, bile-pigment never occurs in the feces in health ; it is present when there is catarrh of the small intestine. Blood-pigment is usually in the form of hæmatin. As might be expected, ptomaines have been obtained from the feces of certain diseases caused by fungi.

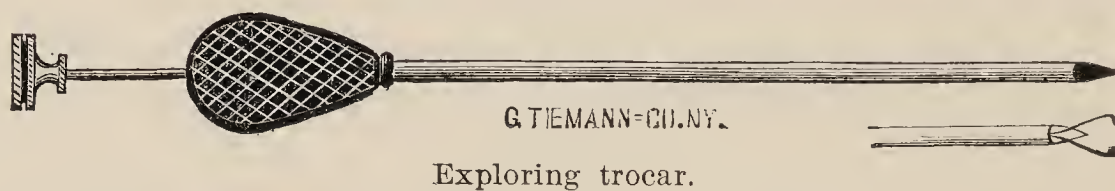
CHAPTER XLV.

EXPLORATORY PUNCTURE.

The presence or absence of fluids in the natural cavities of the body, as the pericardium, the pleura, the abdomen, or the gall-bladder, must frequently be ascertained by means of puncture or aspiration. The fluid is secured at the same time by the puncture for examination. The fluid of tumors or cysts is likewise withdrawn to complete a diagnosis by determining its chemical, microscopical, or bacteriological character. Certain rules of procedure are necessary, and, as they are common to the method in whatsoever situation employed, may be considered in this section.

The Instruments. If it is the desire of the observer to determine the presence of fluid, an ordinary grooved needle may be used. If, however, fluid is to be obtained for examination, a syringe or aspirator must be used. An ordinary hypodermatic syringe, or the syringe of Pravaz may be used if the needles are sufficiently long. A special aspirator made

FIG. 268.



Exploring trocar.

for diagnosis by instrument-makers is the best. The needles are sufficiently long; the barrel large enough to hold sufficient fluid for any method of examination. If the diagnosis is to be followed by aspiration, the apparatus of Dieulafoy, or any equally perfect apparatus, may be used at once. An exploring trocar is used for the removal of pieces of solid tissue as from muscle or from tumors of the lung.

Preparation of Instruments. The instruments should be sterilized in a steam sterilizer or boiled. This does not apply to the needles alone, but every portion of the instrument should be cleansed, because, for instance, the contents of the barrel of the syringe pass through the needle. After sterilization they should be carried to the patient in sterilized test-tubes plugged with cotton-wool. When not in use, the needles should be kept in absolute alcohol and the syringe in carbolic acid solution, 1 : 20. Before using, the carbolic acid should be washed from the syringe and needle with boiling water; they are then to be sterilized as described. Unless the carbolic acid is removed from the syringe, its presence may serve as an antiseptic or disinfectant, and thus interfere with the culture-tests to which the material drawn is to be subjected.

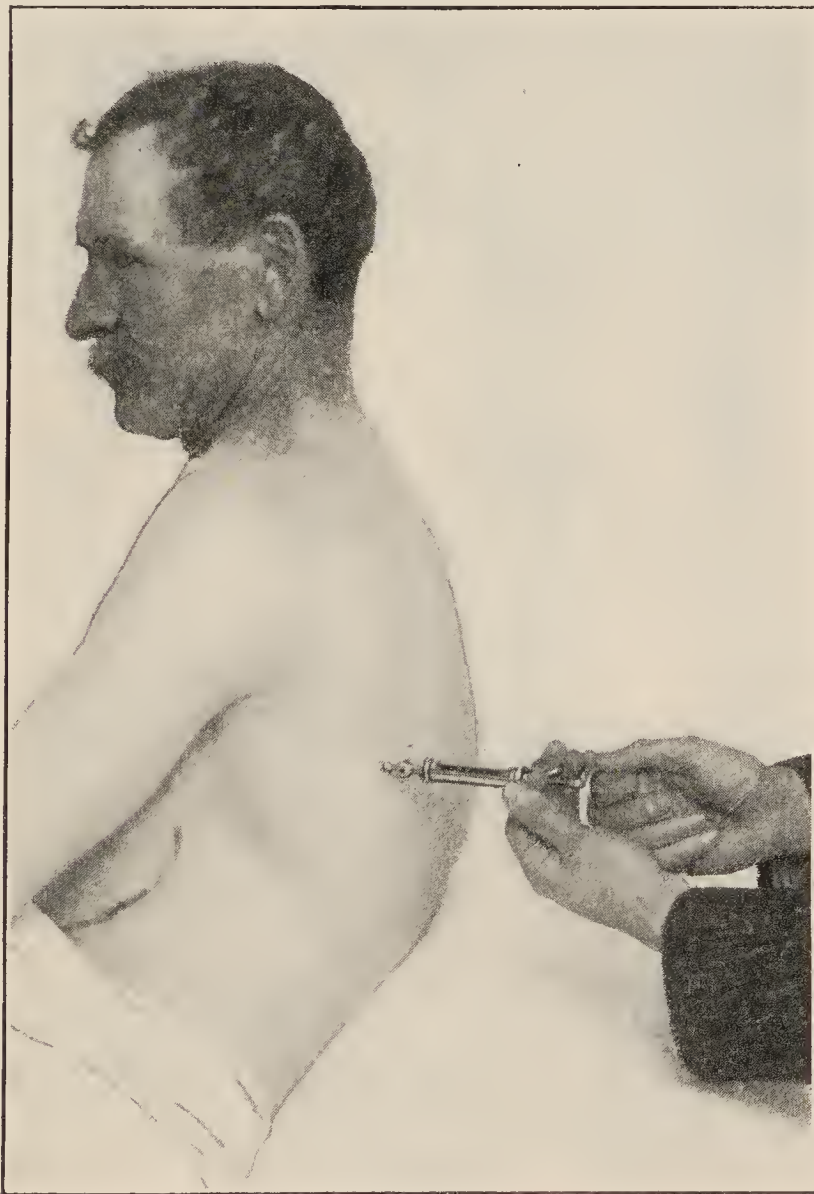
Preparation of Skin. The skin should first be cleansed with green-soap and water, then with alcohol and ether, then with a solution of car-

bolic acid (1 : 30) or of bichloride of mercury (1 : 1000). After thorough cleansing, the parts should be kept covered with a towel soaked in bichloride solution until the time of operation. At the time of puncture the surface should be anæsthetized by ethyl chloride, the rhigolene spray, by ice and salt, or in adults by the Schleich method of subcutaneous anæsthesia. Care must be taken, if the patient is aged or poorly nourished, or the skin œdematous, not to carry the freezing process too far on account of the danger of local gangrene.

The Point of Puncture. The points selected for aspiration depend upon the cavity to be explored or the situation of the cyst.

The Pleura. To withdraw fluid within the pleura, it is best to select a point for aspiration in one of the lower interspaces of the chest, because the fluid is more likely to accumulate in this position and because complete aspiration can there be performed if necessary. The sixth or

FIG. 269.



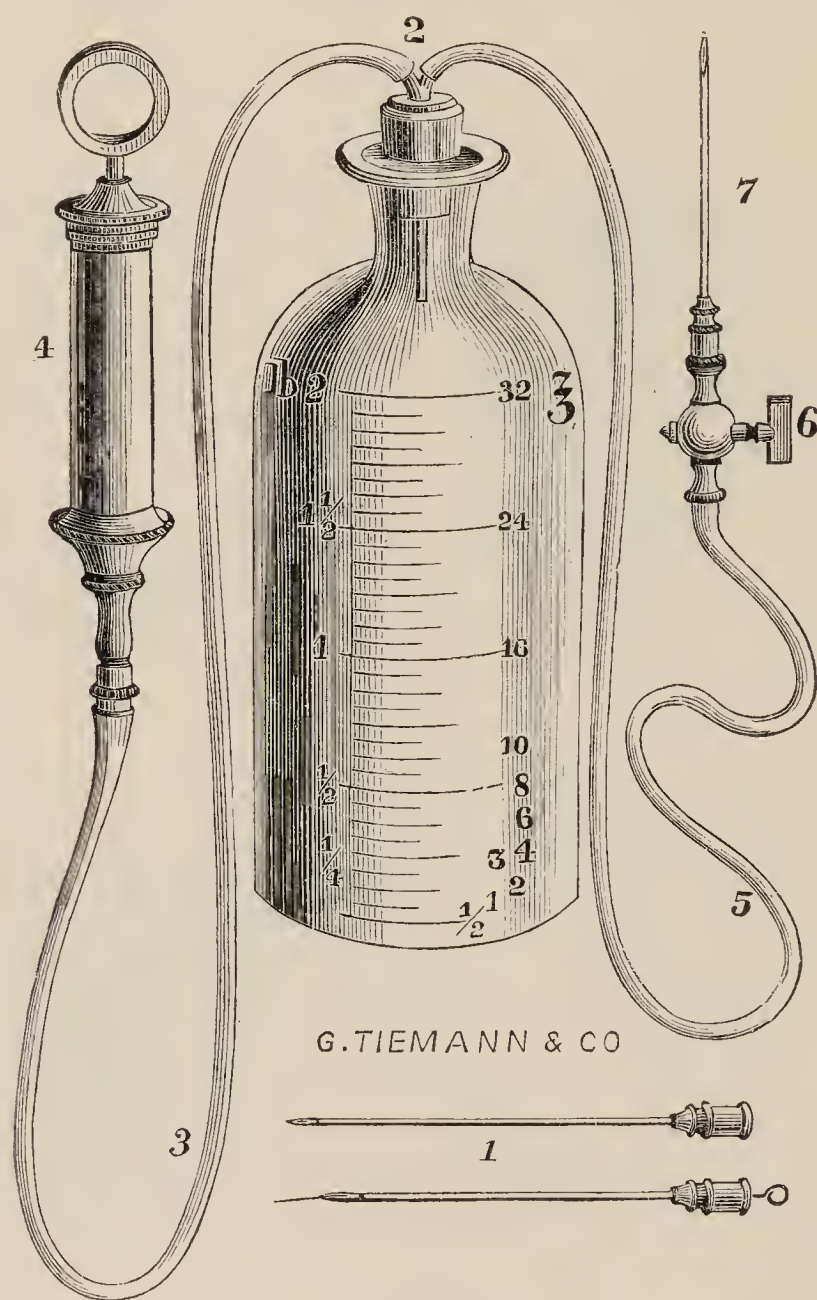
Aspiration of a pleural effusion.

seventh interspace in the anterior axillary line, or the eighth or ninth interspace in the posterior axillary or scapular line, may be selected. On the right side the upper interspace of the two should be chosen on account of the position of the liver. If the contents tend to point or break out at any particular spot on the surface of the chest, the puncture may be made in this area. In suspected loculated empyema or effusions,

the point of puncture should be at the site of greatest dulness and least fremitus.

The Pericardium. For aspiration of the pericardium three points of election have been recommended: first, the usual position of the apex-beat, in the fifth interspace, inside of the midclavicular line; second, the space between the ensiform cartilage and the left seventh cartilage, the point advised by Roberts; third, Rotch has tapped the fifth right interspace a number of times on the cadaver, and thinks that this situation is a proper one in the living subject. The writer has aspirated the pericardium in several instances inside of the normal position of the apex. Care must be taken to insert the needle slowly and with the point directed downward and toward the left axilla when this position is selected.

FIG. 270.

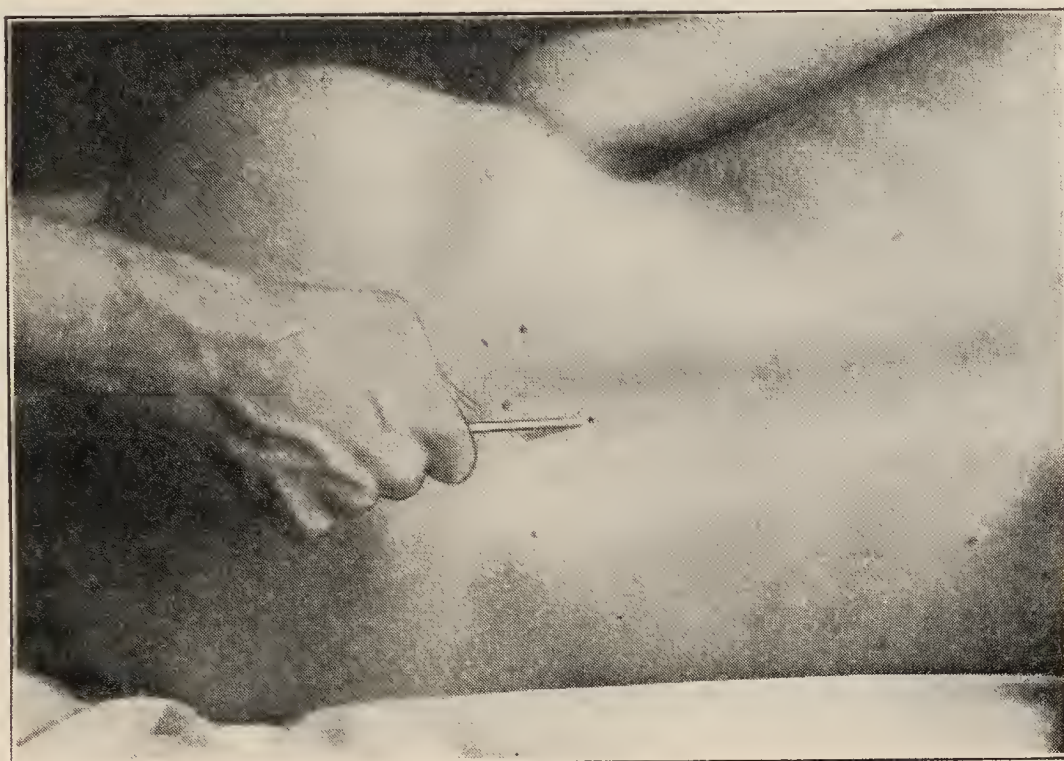


Aspirator.

The Abdomen. It should be remembered that no attempts at puncturing the abdomen should be made if pus is suspected, unless the surgeon is ready to perform laparotomy at once. Indeed, an exploratory operation is performed with so little detriment to the patient by modern surgeons that, on the whole, it should be advocated instead of puncture. There are times, however, when the latter must be resorted to. The writer has performed it in a number of instances without any

danger having ever arisen—always refusing to do so in cases in which pus was probably present in the peritoneal cavity, in the case of tumors or of organs the seat of suppuration. Explorations of this character are probably more feasible in connection with diseases of the *liver*. It does not appear to be harmful to insert needles into that organ, and valuable information is often gained thereby.

FIG. 271.



Lumbar puncture.

The median line should be selected for the puncture in aspiration of the *abdomen*, to determine the character of the peritoneal contents. The bladder must be emptied and a point midway between the umbilicus and pubes selected.

The Vertebral Canal. Spinal or Lumbar Puncture. Proposed by Quincke, the procedure has been carried out by many clinicians and has proved to be a means of establishing a diagnosis. Cerebral lesions are diagnosed and intracranial pressure relieved because of the continuity of the spaces in the brain and the spinal canal. (See Cerebrospinal Meningitis.)

METHOD. The patient should lie on the right side, with the knees drawn up and the left shoulder turned forward. The puncture is made with an antitoxin needle or the needle of a large hypodermatic syringe, which may then be used to withdraw the fluid. The syringe itself may be removed and the fluid allowed to ooze through the needle drop by drop. A needle 4 cm. in length and 1 mm. in diameter is suitable for infants; a longer needle for children over ten years and for adults. Ordinarily the puncture is made between the third and fourth lumbar vertebrae to one side or the other of the median line. The point at which the needle enters the skin should be at the level of the lower end of the spinous process of the fourth lumbar vertebra, one-half inch to one side. The position of the fourth lumbar vertebra may be determined in one of three ways: (1) By counting carefully from the *vertebra prominens*—this

is difficult unless the patient is emaciated, and even then there may be trouble in distinguishing the individual spines of the dorsal vertebræ. The attachment of the twelfth rib can usually be made out, and the spine below it corresponds to the twelfth dorsal vertebra, and is almost immediately

FIG. 272.



Lumbar puncture—introducing the trocar.

above the body of the first lumbar vertebra. (2) The method of Jacoby, which depends upon the fact that a line drawn through the crests of the ilia across the back intersects the spinous process of the fourth lumbar vertebra. (3) The spinous process of the fifth lumbar vertebra is con-

FIG. 273.



Lumbar puncture—introducing the trocar.

siderably more prominent than the spinous processes of the sacrum, and this peculiarity may be utilized as a check upon the other two methods. Practically in all cases all three methods should be employed. A mark on the skin may be made with an indelible pencil or with a soft lead

pencil. As the former is soluble in the alcohol and ether used in preparing the skin, the latter is preferable.

TECHNIC. The technic is as follows: The patient may be either sitting up or lying upon the side. The former position can only be used in patients who are conscious and not suffering from severe disease, as, for example, in cases of brain tumor and chronic hydrocephalus. The patient sits preferably cross-wise upon a narrow operating-table; the body is bent well forward, separating the spines of the vertebra as much as possible. The latter position should be used if the patient is seriously ill. The patient should lie upon the side with the thighs flexed upon the abdomen and the thorax bent over the knees. The site of the puncture should be surrounded by sterile towels. The point of the needle is placed directly over the mark upon the skin; the needle is held at an angle of 45 degrees to the surface of the back, and inclined slightly toward the median line. It is best held—if unprovided with a handle—with the blunt end resting against the palm of the hand, and the shaft grasped between the index and the middle finger and thumb. If it is provided with a handle, the shaft should lie over the index finger, against which it is firmly pressed with the thumb, and the handle is steadied against the palm by the remaining three fingers. It should be thrust forward steadily and slowly, and not turned or twisted. In the great majority of instances, if the patient's back is well arched and the needle is thrust in the direction described, it will enter the spinal canal without difficulty. When it has been inserted between $2\frac{1}{2}$ and $3\frac{1}{2}$ inches, if it has not met with obstruction, it can be assumed that it is in the lumen of the canal and the mandril may be withdrawn. It should not be thrust in farther than this for fear of injuring the point of the needle against the anterior surface of the spinal canal. As soon as the mandril has been withdrawn, ordinarily two or three drops of clear or bloody fluid will flow from the tube. The metal plug should then be inserted into the end of the needle, the zero point on the glass brought as nearly as possible on a level with the spinal puncture, and the intraspinal pressure determined. The fluid can then be permitted to flow into prepared receptacles until it begins to drop very slowly; the needle should then be withdrawn and the opening sealed with a little cotton and collodion.

Certain *modifications* may be employed. A preliminary incision may be made through the skin at the site of the puncture. This should be sufficiently large to admit the needle without touching the skin on either side. It diminishes the liability to infection of the spinal canal by germs carried with the needle from the surface of the skin, but heals more slowly than the simple puncture wound. Infection practically never follows a simple puncture. The puncture may be made in any of the other inter-lumbar spaces where the difficulties are not much greater.

Of the *accidents* that may happen during the operation, the most important is the faulty direction of the needle, so that instead of finding the intervertebral space it strikes upon the arch of the lumbar vertebra. If this occurs, it is only necessary to withdraw the needle a short distance, alter its direction slightly, and thrust again. Usually the direction of the needle is too nearly perpendicular to the plane of the body, in which

case the butt should be slightly lowered. There is considerable variation in the distance between the interspaces in different individuals; in some they are so close together that it is impossible to insert a needle of ordinary size, and the operation becomes futile. If the cerebrospinal pressure is low, and the needle is very dull or has become dull by striking against a bone, it may be difficult to pierce the spinal membranes. When this occurs, of course no fluid is obtained. Sometimes if the needle is thrust in too forcibly and strikes against either the arch of the vertebra or the anterior wall of the spinal canal, it may be bent or broken. If the former occurs, the withdrawal of the needle may be very difficult; if the latter, the piece will probably remain within the spinal canal, where it may give rise to symptoms of irritation. If the needle, in piercing the canal, wounds one of the large veins, the spinal fluid will be bloody, and this may render its subsequent examination unsatisfactory. If it presses against one of the roots of the cauda equina, it may cause pain or twitching of the muscles of the extremities. Sometimes pseudomembrane with pus or gelatinous fluid fills the spinal canal, and hence no liquid is withdrawn. Adhesions may cause the fluid to be loculated.

Sometimes when the needle has been inserted and the spinal fluid has flowed freely, it may suddenly stop. This is due to some obstruction, usually a nerve-root, pressing against the end of the needle. It can usually be dislodged by reinserting the mandril. The cerebrospinal fluid should flow out rather slowly, preferably drop by drop. If it comes in a forcible stream, its flow should be checked either by pinching the rubber tube, or, if the patient is sitting up, having him lie down. If during the operation the patient should complain of headache, dizziness, or nausea, or if he groans, the operation should be interrupted at once and the patient placed either horizontal or with the lower portion of the body slightly elevated.

Data Obtained from Spinal Puncture. The normal pressure of the cerebrospinal fluid when the puncture is made below the conus terminalis and the patient is upright, is between 40 and 100 mm. If the patient is lying down, the pressure ranges between zero and 40 mm. If the pressure is above 150 mm. with the patient in the upright position, it is distinctly pathological. In some cases it may even reach 800 mm. Increase in the cerebrospinal pressure indicates irritation of the central nervous system, due to the presence of a tumor or to some toxic substance in the blood, as for example in uræmia and lead-poisoning, or to some inflammatory process in the cerebrospinal system, particularly meningitis.

Chemical Examination of the Cerebrospinal Fluid. Ordinarily the cerebrospinal fluid contains a mere trace of albumin. Under pathological conditions it may contain as much as 5 to 10 per cent., particularly in meningitis and in brain tumors. If the fluid is bloody, the percentage of albumin may be considerably increased without indicating a pathological change. It contains a substance—pyrocatechin—that reduces Fehling's solution. This ordinarily disappears a few hours after the fluid has been withdrawn. Sugar is said to be present in cases of brain tumor, but not in meningitis.

Histological Examination. The histological examination comprises a careful enumeration of the cells that may be present, and staining for various forms of bacteria. The fluid should be centrifugated, and the sediment spread upon cover-glasses and stained by the usual methods for blood and bacteria. If there is a considerable proportion of large endothelial cells, the rise in pressure is probably non-inflammatory. If the polymorphonuclear forms are in excess, acute cerebrospinal meningitis is probably present. If the mononuclear forms of leucocytes predominate, the infection is presumably tuberculous. A diagnosis based merely upon the cytological examination can not be considered certain. The discovery of bacteria is of more importance. Tubercle bacilli are sometimes found. The meningococcus can be recognized by its form, by the fact that it is intracellular, that several micro-organisms are found in a single cell, and that they stain with unequal intensity. The other forms of micro-organisms are usually found in the fluid. If there is reason to suspect an inflammatory process, cultures and inoculations should be made.

Other methods of obtaining the cerebrospinal fluid, such as puncture of the lateral ventricles of the brain through an opening in the skull, or tapping the fourth ventricle through the intervertebral space in the upper cervical region, are really surgical operations.

Chemical Examination. *Character of fluid.* The fluid is usually clear and limpid in tuberculous meningitis. In other forms of meningitis it is cloudy and turbid. It is purulent in leptomeningitis. Blood may be found in hemorrhage into the lateral ventricles.

Bacteriological Examinations. Cover-glass preparations are made of the fluid, and cultivations taken at once. Streptococci, staphylococci, the pneumococcus, and the meningococcus (*Diplococcus intracellularis*) may be detected in purulent meningitis. Tubercle bacilli have been found in tuberculous meningitis, especially after sedimentation. After the fluid has been twenty-four hours in a conical glass the fine clot which forms should be examined for bacilli. The absence of bacilli does not exclude tuberculosis. The positive result, however, is diagnostic.

Inoculation, as in a case by Lafleur, will cause tuberculosis in a guinea-pig, and is diagnostic. A clear fluid does not exclude purulent meningitis; usually, however, the fluid is purulent, turbid, or rich in leucocytes.

Cysts and tumors, with fluid contents, should be punctured over the point which presents externally, and where they are evidently in closest proximity to the external wall.

The Spleen. The spleen has been punctured for therapeutic and diagnostic purposes. If the organ is hard, as in chronic malaria, it may be done without danger; but if it is enlarged and soft, as in infectious diseases, such as typhoid fever, it is hardly justifiable to puncture it, because of the danger of subsequent rupture. Risk attends the puncture of other organs, as the kidney. The writer has seen a serious hemorrhage follow such puncture, and, of course, septic inflammation may arise. Exploratory operation is more suitable for determining its condition.

THE EXAMINATION OF FLUIDS AND DISCHARGES.

While the fluids to be examined can be obtained by exploratory puncture, it sometimes happens that they are discharged spontaneously, as in the case of an empyema.

FIG. 274.



Degenerated carcinomatous tissue removed during "paracentesis abdominis": 1 and 2, specimens of cells found in exudate from same case; 3, multinucleated cells and "cell inclusion" from case of rectal carcinoma.

The following general methods apply to the examination, in whatever way material is obtained. When derived from the natural cavities, the

fluids are known as exudates or transudates. Fluids are also obtained from cysts, but do not require different methods of examination.

The naked-eye appearances are first noted; then microscopical examination with and without staining is resorted to. Chemical examination is also required. Often culture-preparations and inoculations must be resorted to, as in the case of pus or of serous exudation.

The Exudates.

They may be composed of pus, seropus, gangrenous débris, blood, or pure serum or chyle. When pus, seropus, or putrid fluid is withdrawn, it implies absolutely an inflammatory origin. Blood and serum may be associated with inflammation, simple or infectious; but may also point to impediments in the general or lymphatic circulation. Blood or bloody serum is usually of tuberculous or cancerous origin. Its absence does not imply the absence of either disease. A chylous exudate is usually due to obstruction of the lymph-channels.

Purulent Exudates. Pus ranges in color from gray to greenish yellow. It is turbid, of high specific gravity, and alkaline. It varies in consistence. When standing after removal, it separates into two layers; the upper layer is light yellow and transparent, and the lower opaque. Pus may be mixed with blood, and is then reddish brown. (See Abscess of the Liver.) When it has undergone decomposition, it is thin, green, or brownish red, and of a penetrating odor.

Microscopical Examination. **WHITE CORPUSCLES.** If the specimen is fresh, the cells exhibit the movements that are common in leucocytes. If a solution of iodine and potassium iodide is added to them, they change to mahogany color. If the pus is old and the cells are dead, they are shrunken and granular. Enormous giant cells and cells loaded with fat are seen in pus.

RED CORPUSCLES. In fresh pus, red corpuscles are also seen along with blood-pigment or hæmatoidin crystals.

FREE FAT-GLOBULES and FAT-PARTICLES are present.

EPITHELIUM is rarely seen. In the pus from the pleural cavity, if cancer is present, the vacuolated *epithelial* and *endothelial* cells sometimes seen in cancer may be observed. (See case of Steele and Girvin.)

Bacteria. Micro-organisms are always detected with the aid of staining methods. (See Chapter XXXVIII., Bacteriological Diagnosis.) The micro-organisms are usually the determining cause of the suppuration. Suppuration, however, may be caused by chemical substances, although this is of rare clinical occurrence. Of the various bacteria found, the micrococci and bacilli are the most numerous. The commonest of these are *Staphylococcus pyogenes aureus* and *Streptococcus pyogenes*; *Amœba dysenterica*, in abscess of the liver and secondary abscess of the pleura and lung. It was found in an abscess of the jaw by Flexner.

THE PYOGENIC BACTERIA. 1. *Staphylococcus Pyogenes Aureus*. This micro-organism is found in acute abscesses and boils, sometimes also in

infectious osteomyelitis and ulcerative endocarditis. In addition to other portals, it may enter the tissue through abrasions or the hair-follicles.

Morphology. In cover-glass preparations they appear as small, round bodies scattered among the pus-cells, rarely within them, single, in pairs or in clusters. They stain readily with the basic aniline dyes. (See Fig. 275.)

FIG. 275.

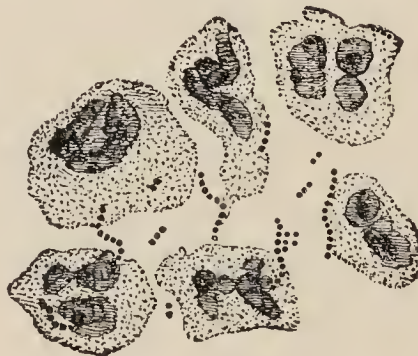
Pus with staphylococcus. $\times 800$. (FLÜGGE.)

2. *Staphylococcus Pyogenes Albus*. It is also found in acute abscesses, but less often than the “aureus,” and is less virulent. It is morphologically identical with the “aureus,” but develops no pigment. The surface cultures are milky white, and the mass at the bottom of the liquefying gelatin is white.

3. *Staphylococcus epidermidis albus* (Welch) closely simulates *Staphylococcus pyogenes albus*. It is the most common micro-organism on the surface of the body, and is often present in parts of the epidermis too deep for disinfection, save by heat. It is supposed to be the usual cause of “stitch-abscess.”

4. *Streptococcus Pyogenes*. It is found in acute abscesses, *erysipelas*, otitis media, puerperal metritis, *infectious endocarditis*, pseudodiphtheria, scarlatinal angina, and most purulent inflammations of a phlegmonous character. It is the organism most commonly found in inflammations having a spreading tendency.

FIG. 276.

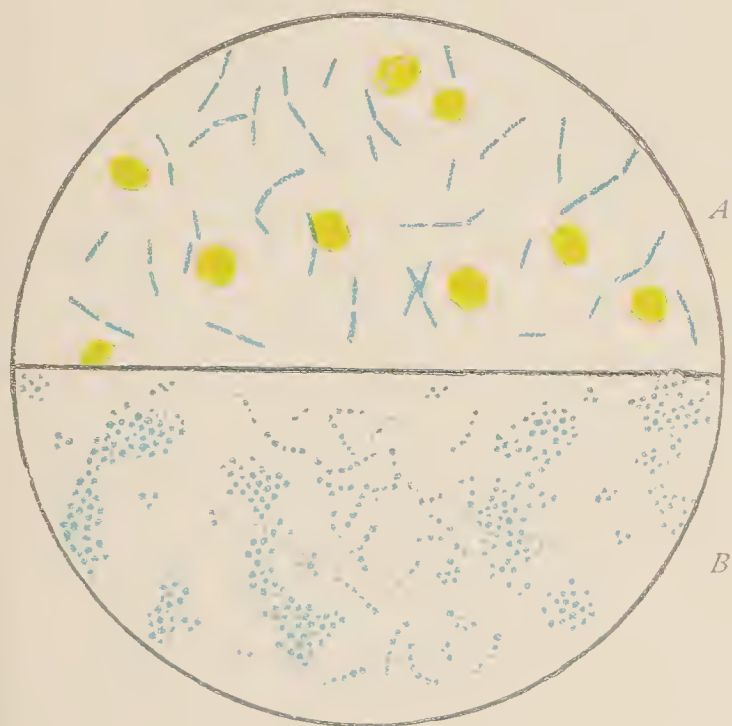
*Streptococcus pyogenes* in pus. $\times 800$. (FLÜGGE.)

Morphology. Cover-glass preparations show spherical cocci of varying sizes, which form chains of 4 to 20 elements, the chains often forming tangled masses. This organism is stained by the basic anilines or by Gram's method. (See Fig. 276.)

Inoculated, it causes erysipelatous or phlegmonous inflammation.

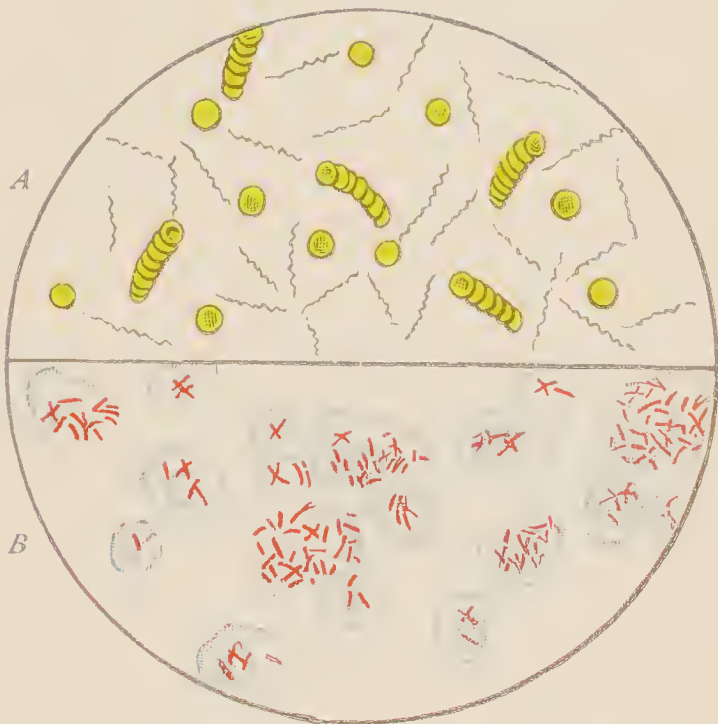
PLATE XIII-c.

FIG. 1.



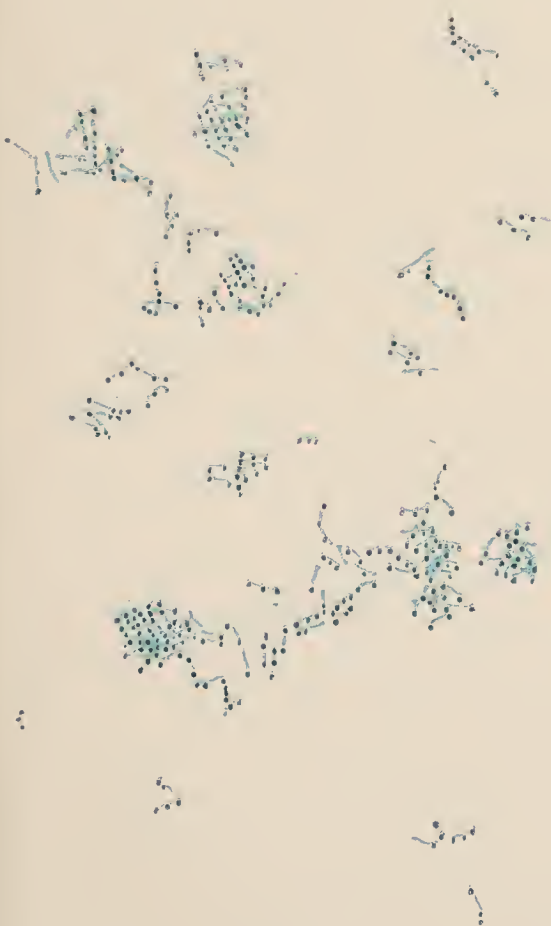
A. Anthrax. B. Streptococcus and Staphylococcus.

FIG. 2.



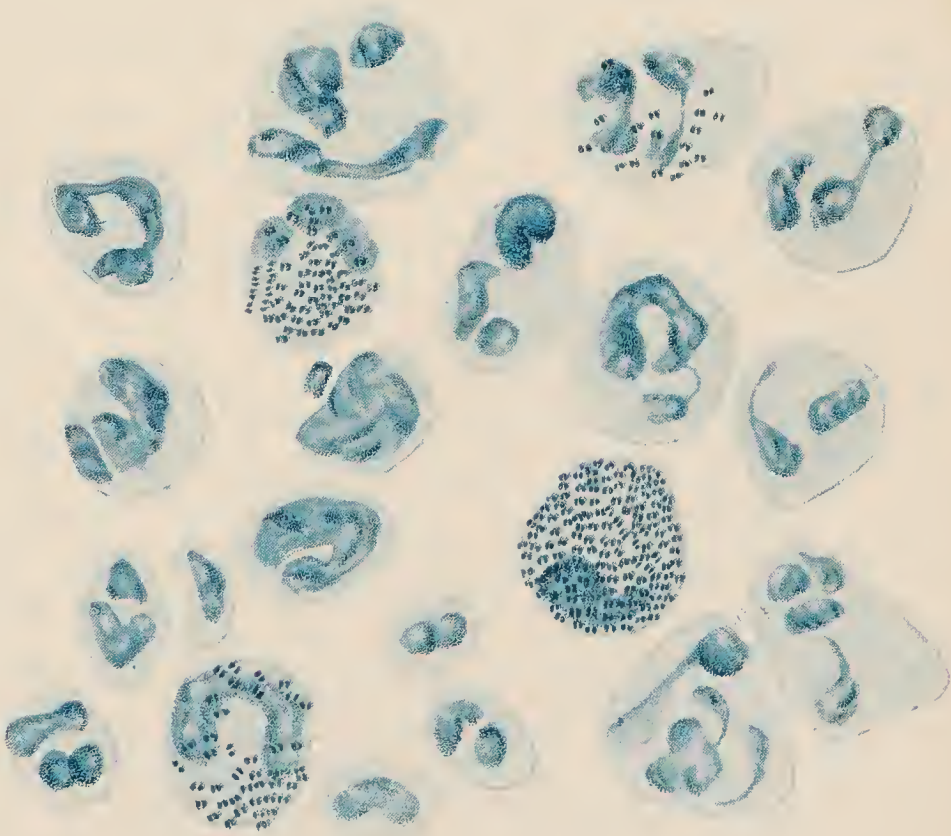
A. Recurrent Spirilla. B. Leprosy.

FIG. 3.



Diphtheria Bacillus.

FIG. 4.



Gonococcus.

5. *The Tubercle Bacillus*. This is seen at times in pus removed from phthisical cavities, and the pus of abscesses, particularly about glands. It may be detected by methods of staining adopted in the examination of the sputum. Pus may be of tubercular origin, and the micro-organisms may not be detected by the usual microscopical methods. Its absence therefore does not imply the absence of tuberculosis. Culture-methods and inoculation should be resorted to, particularly the latter.

6. *The Bacillus of Syphilis*. The pus under these circumstances is usually derived from ulcers or inflammations, or from secretions about the vulva or prepuce. The actual relationship to syphilis has not been demonstrated.

Lustgarten's method is as follows: After immersion for twenty-four hours at the ordinary temperature in the gentian-violet fluid of Koch-Ehrlich, the cover-glass preparation is removed and washed for a few moments with absolute alcohol. It is then placed for ten seconds in a 1 per cent. or 2 per cent. solution of potassium permanganate; a watery solution of pure sulphurous acid is then poured over it, after which it is washed in water. If the preparation still shows its color, it must be reimmersed for a few seconds in the potash solution and then in the sulphurous acid, and again washed with water.

7. *Actinomyces*.

8. *The Bacillus of Glanders*.

9. *The Bacillus of Anthrax*.

10. *The Bacillus of Leprosy*.

11. *The Bacillus of Tetanus*.

12. *The Bacillus of Influenza*. (See Sputum.)

13. *Micrococcus Lanceolatus* or *Pneumococcus*. The pneumococcus is often found in the pus of empyema and pericarditis, whether from the pleural cavity or after it has burrowed from this situation. It occurs in cerebrospinal meningitis. It is easily detected by the usual staining methods (for which see Sputum).

14. *Bacillus Coli Communis*. This is found more commonly in infections within the abdominal cavity. (See Feces.)

15. *The Gonococcus*. It is constantly present in virulent gonorrhœal pus, usually within the pus-cell or attached to the surface of epithelial cells. *Morphology*: Micrococci, usually joined in pairs or groups of four flattened and separated, when stained, by an unstained intracellular space. Stains easily with aniline—not by Gram's method. No other coccus is of the same shape, nor present at the same time within the cells, except the meningococcus, which is of the same shape, likewise intracellular, and is decolorized by Gram's method. (See Plate XIII., C, Fig. 4.) It grows more abundantly and readily, however.

PROTOZOA IN THE PUS. Cercomonads have been observed in the pus obtained from an empyema, and are probably derived from the lungs. Flexner has found *Amœba dysenteriae* in the pus from an abscess of the jaw. It is found in abscess of the liver and secondary abscess of the lung. (See Sputum and Feces.)

VERMES. *Filaria* have been found in abscess of the liver. The pus from a hydatid cyst contains membrane and hooklets.

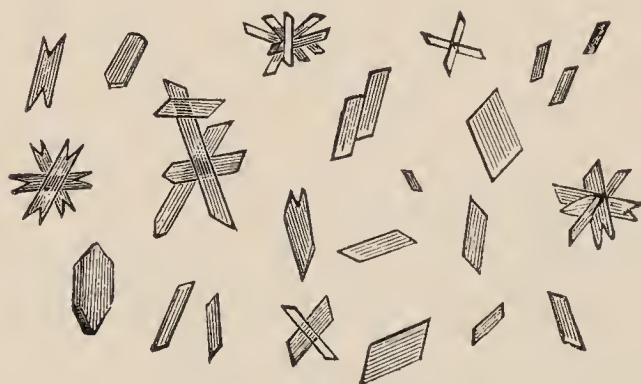
CRYSTALS. Crystals of *cholesterin* are found in the pus from cold abscesses, suppurating ovarian cysts, and fetid discharges. They are similar to the crystals described under sputum.

Hæmatoidin crystals indicate a previous hemorrhage; they are most frequent in suppurating hydatid cysts. (See Fig. 277.) *Fatty needles* are found in old pus and gangrenous exudates. (See Fig. 278.) *Triple phosphates* are frequently seen in pus, and are of the same appearance as the phosphates in the urine. The carbonates and phosphates are seen in fetid pus.

Chemical Examination of Pus. This does not yield any information of diagnostic value.

Serum-albumin, globulin, and peptone are detected by methods employed in the examination of urine. Fresh pus contains sugar. After being boiled with an equal weight of sodium sulphate and filtered, the filtrate is examined by the reagents used in the examination of urine for sugar. Pus also contains bile-pigments and biliary acids, cholesterin, and salts of sodium, and the fatty acids in jaundice. Von Jaksch has found acetone in pleural exudates.

FIG. 277.



Rhombic crystals of hæmin. (CHARLES.)

FIG. 278.



Pus from putrid empyema. (Eye-piece III., obj. 8, A. Reichert.) Shrunken leucocytes. Fat-crystals. (VON JAKSCH.)

Seropurulent Exudates. They resemble purulent discharges, chemically and morphologically. They point to antecedent inflammation.

Putrid Exudates. The exudates are brown or brownish green in color. The odor is penetrating and offensive. They are usually alkaline in reaction. On *microscopical examination* old leucocytes and crystals of fat, cholesterin, and hæmatoidin are seen; fission-fungi of various forms are also seen. (See Figs. 277 and 278.)

Hemorrhagic Exudates. Hemorrhagic exudates contain *red blood-corpuscles* and *hæmoglobin* in large amount. *Fatty endothelial cells* are found. Quinke states that when the glycogen-reaction is shown, if the fluid is from the pleura, carcinoma is probably present. A positive diagnosis depends upon the discovery of the epithelial cells (see page 691) which are seen in cases of cancer. Hemorrhagic exudates in the pleura are due most frequently to cancer, to tubercle, or to scurvy. To determine its exact nature as tubercle, inoculation and cultures are sometimes necessary.

Serous Exudates. The fluid is clear and light yellow or straw-

colored. On standing a white fibrinous clot is deposited. On *microscopical examination*, red blood-corpuscles, leucocytes, fatty globules, and endothelial cells are found. They may be bunched in groups or scattered about. The micro-organisms, if present, are detected with difficulty. If ulcerating tuberculosis of the pleura is present, the bacillus may be found, but tuberculous pleurisy may exist without ulceration, and hence the fluid does not contain the bacillus. Cholesterin crystals are found in old serum. On *chemical examination* the fluid contains more than 3 per cent. of serum-albumin and globulin; peptone is absent in pleural exudations; sugar in small amount and acetone are found. The specific gravity of the fluid is above 1018.

Ascites. Evidence of the nature of an ascitic fluid—that is, whether it is an exudate or a transudate—may be obtained from the specific gravity and the percentage of albumin that it contains. The transudate that occurs in cachectic conditions, such as grave anæmias, chronic nephritis, amyloid disease, carcinomatosis, etc., has a specific gravity of 1010 or less, and contains 1 per cent. or less of albumin. The transudate that occurs in obstructive conditions in the liver and the portal circulation, such as cirrhosis of the liver, pylephlebitis, etc., has a specific gravity of from 1010 to 1014, and contains from 1 to 3 per cent. of albumin. The exudate that occurs in local inflammatory conditions of the peritoneum, tuberculous and cancerous peritonitis, etc., has a specific gravity of from 1017 to 1020 or higher, and contains from 4 to 6 per cent. of albumin. The fluid contained within an ovarian cyst has a specific gravity, as a rule, of 1025 or higher.

The percentage of albumin in the ascitic fluid may be calculated according to the formula of Reuss, which is said to be subject to an error of less than 0.25 per cent. According to this formula, the percentage of albumin equals three-eighths of the last two figures of the specific gravity (the specific gravity minus 1000), minus 2.8. Thus, should the specific gravity of the ascitic fluid be 1016, the percentage of albumin may be calculated, as follows: $\frac{3}{8}$ of 16 equals 6; 6 minus 2.8 equals 3.2, which is the percentage of albumin.

Chylous Exudates. True chyle is found in fluids of low specific gravity. Such an effusion is rich in fat and is due to leakage of lymphatics into the peritoneal cavity. It is known as a chylous effusion. Chyliform effusion is a term applied to the second variety of effusions mentioned in this section. The fluid has the property of chyle. Sometimes in peritoneal exudation, particularly if the patient has been upon a milk diet, the fluid contains fatty matter, which gives it a milky appearance. The same character of fluid is seen in obstruction of the thoracic duct.

Special Effusions. Effusions into the Pleura. The effusion may be serous, serosanguinolent, bloody, or purulent. It is of the greatest importance to distinguish the various forms of infection. Bacteriological examination is necessary. In purulent exudation, if micro-organisms are absent (staphylococcus and streptococcus), it is probably tuberculous; serofibrinous exudations are usually free from fungi. When *Micrococcus lanceolatus* is found, it is of favorable prognostic omen.

To distinguish the *effusion of inflammation* from that of *transudation* (obstruction) the specific gravity is suggestive. In the inflammatory effusions the specific gravity is high; they also contain a large amount of fibrin and more than 3 per cent. of albumin.

Transudates.

This class of fluids is serous, bloody, or chylous. The specific gravity is lower than in inflammatory effusion. The color is light and the reaction usually alkaline. On microscopical examination but little is found. In pleuritic effusions there may be considerable endothelium, which, if mixed with blood, may be due to carcinoma. Serum contains albumin and sugar, the former in great excess. Peptone is always absent. The fluid coagulates with difficulty on boiling.

Runeberg¹ lays stress upon the diagnostic importance of the amount of albumin in pathological transudates and exudates. His experience warrants the following statements:

1. Inflammatory processes, 4 to 6 per cent. of albumin.
2. Venous stasis, 1 to 3 per cent. of albumin.
3. Marked hydræmic conditions, as in amyloid degeneration or nephritis, 0.1 to 0.5 per cent.
4. Combination of two or three of the above causes, 0.2 to 6 per cent.

In old transudates due to venous stasis, even without inflammatory complications, the percentage of albumin may be high.

Contents of Cysts.

In aspiration of the abdomen and of the pleura, cysts are sometimes evacuated, the nature of which is often determined by an examination of the fluid. It is within the province of this work to discuss hydatid cysts, pancreatic cysts, and the cystic kidney. As tumors of the ovary so frequently resemble tumors in other situations, it is well also to discuss in this section the nature of the fluid withdrawn from them.

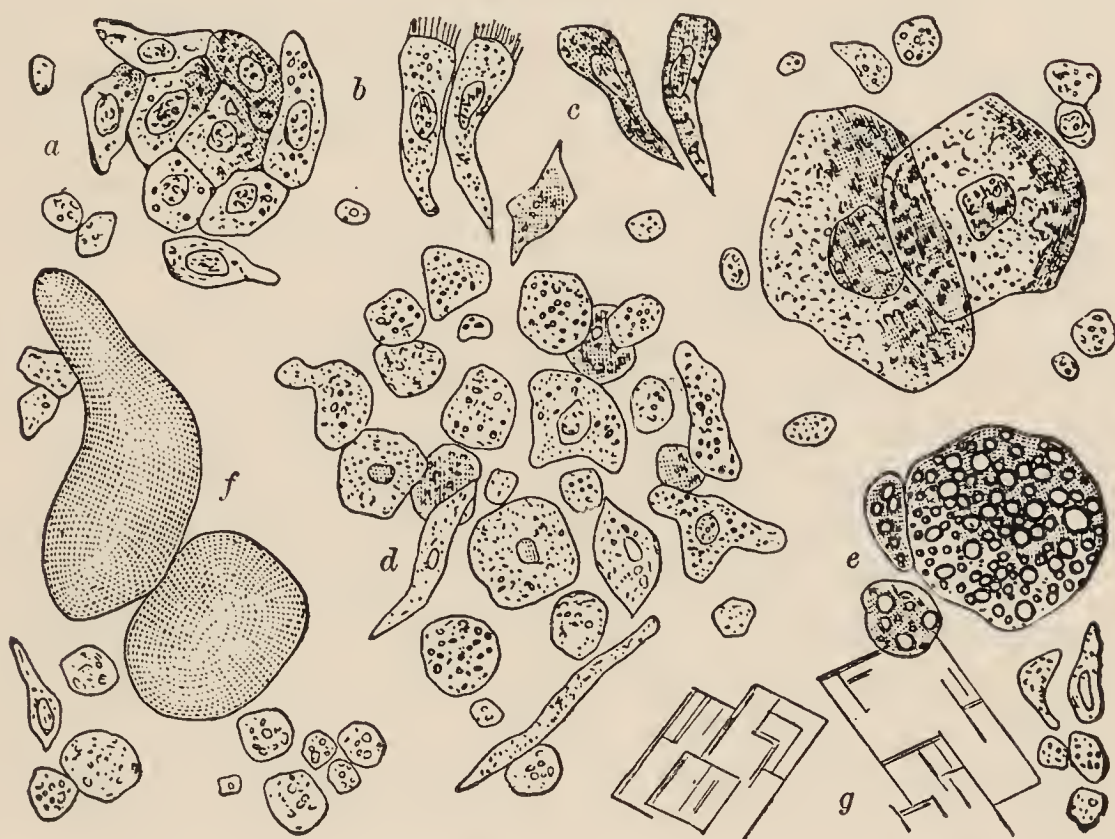
Hydatid Cysts. The fluid of hydatid cysts is clear, alkaline, and of a specific gravity of 1010. It contains sodium chloride in excess, grape-sugar in small amount, and very little if any albumin. On microscopical examination hooklets are found, as in the sputum from hydatid cyst of the lung, as well as portions of membrane. The membrane is recognized by its peculiar transverse striation and the granular appearance of its inner surface. The heads or scolices are sometimes found. Two circles of hooklets and four disks on the anterior aspect cross the head, which is separated from the posterior part by an annular constriction. (See Sputum and Feces.) If suppuration has taken place, the original nature of the cyst can not be made out unless hooklets are found. After the fluid has been standing in a conical glass vessel the bodies may be found in the sediment.

Ovarian Cysts. The fluid from an ovarian cyst is of high specific gravity, 1026, of alkaline reaction, contains but a small amount of

¹ Runeberg, J. W., "On the Diagnostic Importance of the Amount of Albumin in Pathological Transudates and Exudates." *Berliner klin. Wochenschrift*, 1897, No. 33.

albumin, and does not coagulate. On microscopical examination various forms of epithelial cells are seen, colloid bodies, and cholesterin crystals. If hemorrhage has taken place in the cyst, the color of the fluid is correspondingly changed, and besides the squamous, columnar, and ciliated varieties, some epithelium in the stage of fatty degeneration and red and white blood-corpuscles are seen. In colloid cysts the usual concretions are found. (See Fig. 279.)

FIG. 279.



Contents of an ovarian cyst. (Eye-piece III., obj. 8, A. Reichert.) *a*, squamous epithelial cells; *b*, ciliated epithelial cells; *c*, columnar epithelial cells; *d*, various forms of epithelial cells; *e*, fatty squamous epithelial cells; *f*, colloid bodies; *g*, cholesterin crystals. (VON JAKSCH.)

In *dermoid* cysts, in addition to the above, squamous epithelium, hairs, and fatty, hæmatoidin, and cholesterin crystals are detected. *Ovarian fluid* contains albumin and methæmoglobin, or paralbumin. The latter is detected by mixing a portion of the fluid with three times its bulk of alcohol. It is then allowed to stand for twenty-four hours, when it is filtered. The precipitate is removed and suspended in water. After filtering, the filtrate is seen to be opalescent, and is tested as follows:

1. On boiling no precipitate is formed, but the fluid becomes turbid.
2. There is no change with acetic acid alone.
3. The fluid becomes thick and of a yellowish tint when treated with acetic acid and potassium ferrocyanide.
4. There is a change to a violet color when treated with concentrated sulphuric and acetic acids.

Some observers differ from the above statement in their description of the fluid of an ovarian cyst; all agree as to the large number of cell-elements. At one time it was thought that the fluid contained a special cell, but this view has been abandoned. In rare cases the specific gravity may be lower than that of the fluid of ordinary ascites. A fluid of low specific gravity, with a small amount of albumin, is said to be characteristic of a cyst of the broad ligament.

Cystic Kidney. The fluid from a cystic kidney can be recognized by

the properties it derives from the renal secretion. Urea and uric acid in large amounts point to its true source. Renal epithelium is of the greatest diagnostic value. (See Urine.) If epithelium from the urinary tubules can be detected after the fluid has settled, the diagnosis is absolute. (See Hydronephrosis.) It must not be forgotten that both urea and uric acid may be found in other cysts, as in those of the ovary, if they communicate with the urinary tract.

Pancreatic Cysts. The fluid from cysts of the pancreas is of a specific gravity of 1012, but may be as high as 1028. It contains cholesterin crystals in abundance, and blood or pigment. Serum-albumin is present, but metalbumin is not found. Three diastatic ferments are present:

1. If on examination for sugar the latter is found to be a maltose, its presence is of diagnostic significance.

2. The most pronounced property of pancreatic fluid, and that by which we are enabled to distinguish it from other fluids, is the power of digesting albumin without the presence of an acid.

Boas¹ developed the method of examination. The fluid is to be added to milk. After the casein is precipitated the biuret test is applied, as follows: Heat the substance with caustic potash and add, drop by drop, a 10 per cent. solution of sulphate of copper. If digested albumin is present, the fluid assumes a reddish-violet color. No other cystic fluid can dissolve albumin in alkaline solution.

It is not necessary that albumin or fibrin should be employed in performing this test, as it is sufficient to add milk to the secretion; when in such cases the casein of the milk is precipitated, and the biuret test is applied to the resulting filtrate, and the test compared with a control-milk from which the casein has been removed (this can be done by adding very dilute acetic acid with constant stirring), the digestive property of the liquid under examination may be with certainty determined. The peptone would not be precipitated with the albumin, and as all albumins give the same reaction as peptone with the biuret test, the albumin should be removed before applying the test. It is removed from the filtrate by a saturated solution of ammonium sulphate. Then test the resulting filtrate with the biuret test, and compare with the control test as above.

3. The pancreatic fluid also emulsifies fats. In large cysts, however, particularly if of long standing, the physiological properties of the pancreatic juice are sometimes wanting.² In the case referred to by Boas and reported by Karewski, the protracted duration of the cyst modified the character of the fluid, and hence rendered its nature doubtful. Moreover, in the exploratory puncture the stomach was penetrated. For two reasons the author advises against exploratory puncture. First, the age of the cyst is not known, hence an analysis would be misleading. Second,

¹ Deutsche med. Wochenschr., 1890, Bd. xvi., S. 1095.

² In a case operated on by Penrose the analysis of the fluid was as follows: Sp. gr. 1025; reaction slightly alkaline; serum-albumin; no metalbumin; diastatic ferment absent; maltose absent. By Boas' method, power to digest albumin appeared to be great; but when the albumin remaining in the filtrate was removed from the pancreatic fluid, it failed to show that peptone was formed. The method, therefore, appears to be fallacious in this class of cases. The cyst was old, and the fluid no doubt had lost its physiological properties. Cholesterin was present in enormous amount; tyrosin crystals were scarce.

the danger of puncturing other organs is too great. Exploratory laparotomy is preferable.

Cyto-diagnosis. An endeavor has been made to associate certain cells of the blood and tissues found in exudates with certain clinical conditions. Wedal and Ravant have given the most systematic information. The result of their investigations showed that :

I. Tuberculous Affections. Pleurisies and meningitis were associated regularly with an increase of the mononuclear elements of the blood in the exudate—lymphocytosis up to 65 and 100 per cent.

II. Acute Infections. Streptococcus and pneumococcus pleurisies, etc., are accompanied by an increase of the polymorphonuclear blood elements in the exudates.

III. Mechanical Disturbances. The exudate most frequently contains the cells of the serous membrane, endothelial cells either simple in form or polynuclear and vacuolated.

There has been abundant confirmation of the earlier findings, and one may say that a lymphocytic exudate is always suggestive of a primary tuberculous affection. The multinuclear elements may be slightly increased in the first few days, or if secondary infections ensue, or if a tuberculous process is of the nature of an extension from adjacent parts, as pyopneumothorax, but in the end the lymphocytes will predominate.

Quincke, Eichhorst, and others have endeavored to diagnosticate malignant growths from the character of the cells in exudates or secretions. This has been readily done when tumor particles have been present, but can not be relied upon in all cases. The presence of fatty granular cells giving the glycogen reaction has been wrongly considered suggestive. The presence of numerous cells showing mixtures, typical and atypical, is frequently seen in exudates due to malignant irritation and is by some considered diagnostic. If the character of the cells is such that they can be differentiated from endothelial and other cells, a conclusion may be reached, and it is to be noted that the endothelial cells present as a result of simple irritation, only rarely show mitotic figures; they are also, as a rule, larger than other cellular elements. Such cells with mitotic figures may, however, be scant or absent in malignant exudates, and cells showing amitotic division of nucleus may be the only feature.

Infectious Exudates. In one of my cases of ascites studied by Gwyn, small round cells, single or in bunches (suggestive of carcinoma), led to a careful examination of the fluid. No mitoses were seen, but the further presence of regular cylindrical cells in a glandular arrangement, resembling a Lieberkühn crypt, and pieces of tissue enabled me to diagnosticate an intestinal carcinoma. Steele was so fortunate as to make a diagnosis of pleural carcinoma by finding cells in the exudate.

Multinuclear cells, cells with mitotic figures, and with inclusions (Russel's bodies, cancer parasites, etc.), were observed in the stools of a patient who on proctoscopic examination was shown to have a rectal cancer. The sputum of a case with obscure pulmonary symptoms was found to consist so largely of small round mononucleated cells that a suspicion of a new growth was entertained. Autopsy revealed a sarcoma of the lung.

PART II.

SPECIAL DIAGNOSIS.

CHAPTER I.

THE INFECTIONS.

INFECTIONS RECOGNIZED BY THEIR CLINICAL COURSE.

THIS group includes most of the contagious and epidemic eruptive fevers. Their recognition is largely based on the duration of the period of incubation, and upon the mode of onset and clinical course of the respective infection. They are: *typhus fever*, *smallpox*, *varicella*, *scarlet fever*, *measles*, and *rubella*. They are also known as the eruptive fevers, and upon the character of the respective eruption, which bears a definite symptomatic relation to the infection, the diagnosis is, in great part, based.

Typhus Fever.

Typhus fever is an acute contagious disease, occasionally occurring sporadically, and often becoming epidemic in the presence of destitution, filth, over-crowding, and poor ventilation. It is characterized by abrupt onset with chill or with chilliness, a *rapid rise* of temperature, lassitude, headache, and pains in the back and limbs. By the end of the first day the temperature is 104° to 105° F. On the fourth or fifth day a peculiar spotted eruption appears, which at first is macular and subsequently petechial. It is further characterized by adynamia or ataxia, low muttering delirium, a suffused, heavy, drunken expression of countenance, by the absence of local lesions, and by a crisis which occurs on or about the fourteenth day.

Typhus fever is variously known as *ship fever*, *jail fever*, and *camp fever*.

Incubation. The period of incubation is usually about twelve days; it may be five or eight days, or even a shorter time, depending upon the virulence of the poison and the susceptibility of the patient. Malaise may precede by a day or two the onset of the disease.

Invasion. Invasion is characterized by headache, faintness, vertigo, chilliness, or a distinct rigor, pains in the back and thighs, loss of appetite, nausea, constipation, and extreme weakness. The prostration is sometimes so great as to compel the patient to go to bed at once. The

pulse is frequent, 100 or 140, and in grave cases shows a marked tendency to become small, soft, and feeble. The patient is restless and sleepless, and is annoyed by tinnitus. The *expression* of the flushed face is listless and dull.

The Eruption. About the fourth or fifth day the typhus eruption begins to appear. It consists at first of dull-red spots of irregular size and shape. They are most numerous on the covered parts. Moore¹ says they are detected first near the axillæ and on the wrists, then on the sides of the abdomen, afterward on the chest, back, shoulders, thighs, and arms. The skin is also mottled by another crop of maculæ under the skin ("mulberry rash").

When the disease is fully developed, the face is flushed, the conjunctivæ red, the pupils contracted, so as to resemble pin-holes ("ferret eye"), the tongue dry and brown, the teeth covered with sordes, the skin dry, hot, and stinging to the touch. The patient lies upon his back oblivious to his surroundings. Headache has given place to delirium, which may be wild and fierce, but is more commonly low and muttering. There are marked ataxic symptoms—subsultus tendinum, tremors, and picking at the bedclothes. Incontinence of urine and feces sometimes occurs. The breathing is frequent, shallow, and noisy, and the pulse more rapid, soft, and feeble. The macular rash now becomes petechial. The patient is in a typical "typhoid state." The stupor may gradually clear up, or, on the other hand, deepen into coma; or the patient may die from progressive weakening of the heart, with or without pulmonary complications.

In the majority of favorable cases, on or about the fourteenth day, the first sign of recovery is a sound sleep, from which the patient awakes refreshed and rational. The temperature falls with great rapidity and the pulse and respiration improve—a typical crisis has occurred.

Certain *objective phenomena* of the disease require special mention. The eruption is more copious in severe than in mild cases. A dull and livid color is a grave sign. Purpura and hemorrhages are sometimes met with in severe cases. The eruption does not occur in successive crops.

The patient seems to be surrounded by a vapor of a pungent, musty *odor* which is peculiar.

The *heart* early shows the effect of the poison. The impulse is diminished, and the first sound is less distinct. In grave cases, with threatening heart-failure, the sounds are feeble and distant, the impulse imperceptible.

The *pulse* is usually very much more frequent than normal, but may be abnormally slow (50 and even 30 per minute); this is sometimes a bad sign.

The *weak heart* and prostrate position of the patient favor congestion and œdema of the lungs. This condition is common.

Digestive symptoms have already been referred to. Vomiting, tympanites, and diarrhœa are rare, and still more so is intestinal hemorrhage.

The *urine* is scanty and high-colored. Slight albuminuria is common,

¹ Eruptive and Continued Fevers, by J. W. Moore, Dublin, 1892.

and a few casts are found, but distinct nephritis is unusual. Convulsions, when they occur after the first week, are almost always uræmic and almost invariably fatal. They may be due to retention of the urine, as recorded by Stokes and Corrigan.

Duration. The duration of the disease is from six to fifteen days; the average period is twelve to fourteen days. An abortive form is met with in some epidemics, the disease being of a mild type and subsiding at the end of a week. In some cases so large a dose of the poison is absorbed by the patient that he is stricken down in a few hours or a few days. To this form the name "blasting typhus" has been appropriately given. The most important complications are hyperpyrexia, laryngitis, bronchitis, and congestion of the lungs, extreme ataxia or profound adynamia, nephritis, heart-failure, and parotitis or other inflammatory glandular swellings.

Laryngitis with œdema is a very rare but very dangerous complication.

Diagnosis. *H. D.* Hygienic conditions; epidemic exposure.

S. D. Clinical course.

O. D. Eye; eruption; range of temperature.

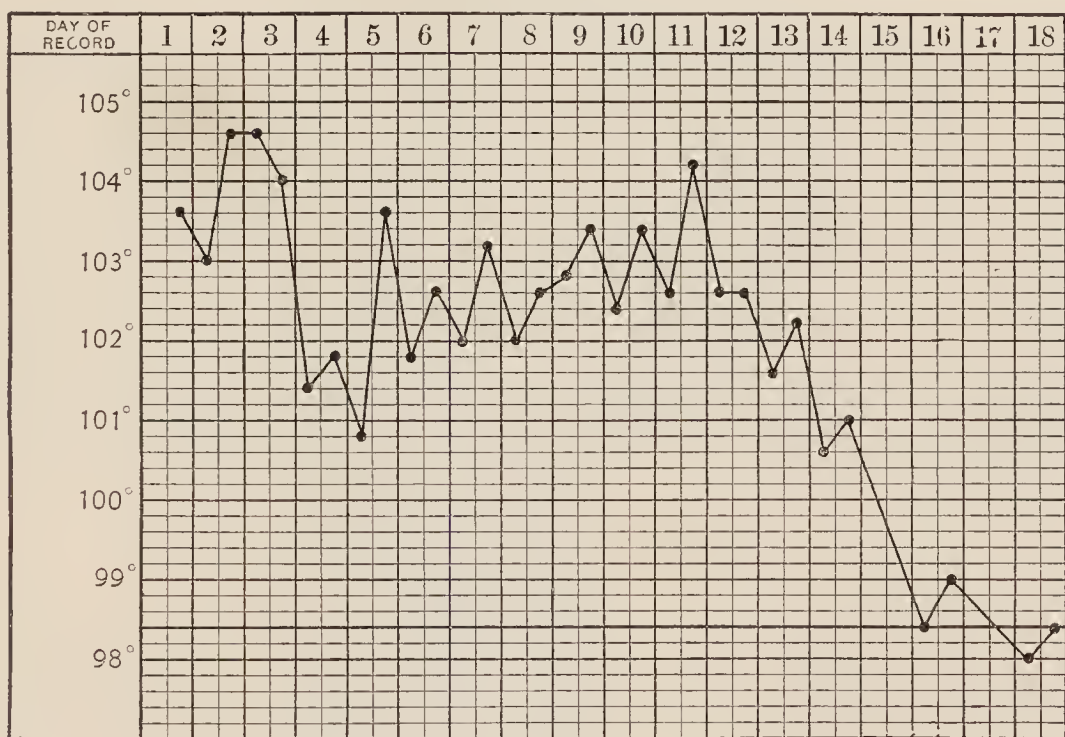
P. D. Negative.

L. D. Negative.

D. D. From cerebrospinal meningitis, uræmia, pneumonia, typhoid fever.

Cerebrospinal fever is distinguished from typhus fever by greater intensity of the headache, by retraction of the head and hyperæsthesia, by greater liability to vomiting, by the presence of leucocytosis, by the result of lumbar puncture, and by the absence of the macular petechial

FIG. 280.



Typhus fever—typical. (DOTY.)

eruption and the drunken, besotted aspect of typhus fever. In cerebrospinal fever the patient suffers with photophobia, and is liable to local palsies of the eye-muscles (strabismus) and to general convulsions. Convulsions do not occur in typhus except from a complicating nephritis or retention of urine.

Uræmia is distinguished from typhus by the preceding history, by the absence of high temperature, and by the presence of œdema of the face or extremities, a history of vomiting or diarrhœa preceding the stupor. The condition of the urine and the absence of eruption are the final tests.

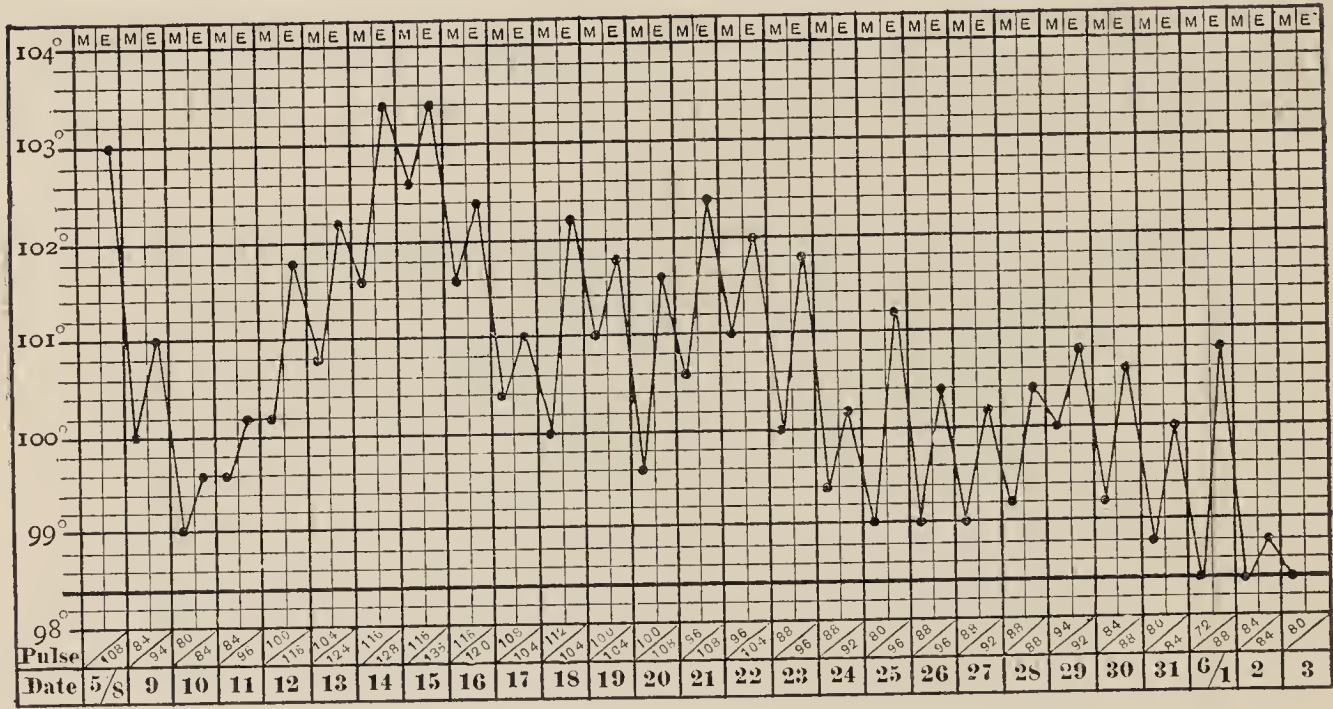
Pneumonia is distinguished by the frequent respiration and relatively slower pulse, and by the local physical signs and absence of eruption.

Typhoid fever is distinguished by its slow onset and marked abdominal symptoms. The eruption of typhus is petechial and comes out on the fourth or fifth day; that of typhoid fever consists of rose spots and appears on the seventh or eighth day. In typhus fever the severe initial chill, the sudden onset, the greater prostration, and the earlier appearance of cerebral symptoms are helpful in distinguishing it from typhoid fever. The serum reaction must be employed.

Variola.

The temperature in variola, or smallpox, pursues a definite course, which renders it of value in the diagnosis. Its sudden rise to an unusual height without local inflammation but with severe backache is significant. Its fall with the appearance of the eruption, followed in two or three days by a secondary rise, is very characteristic.

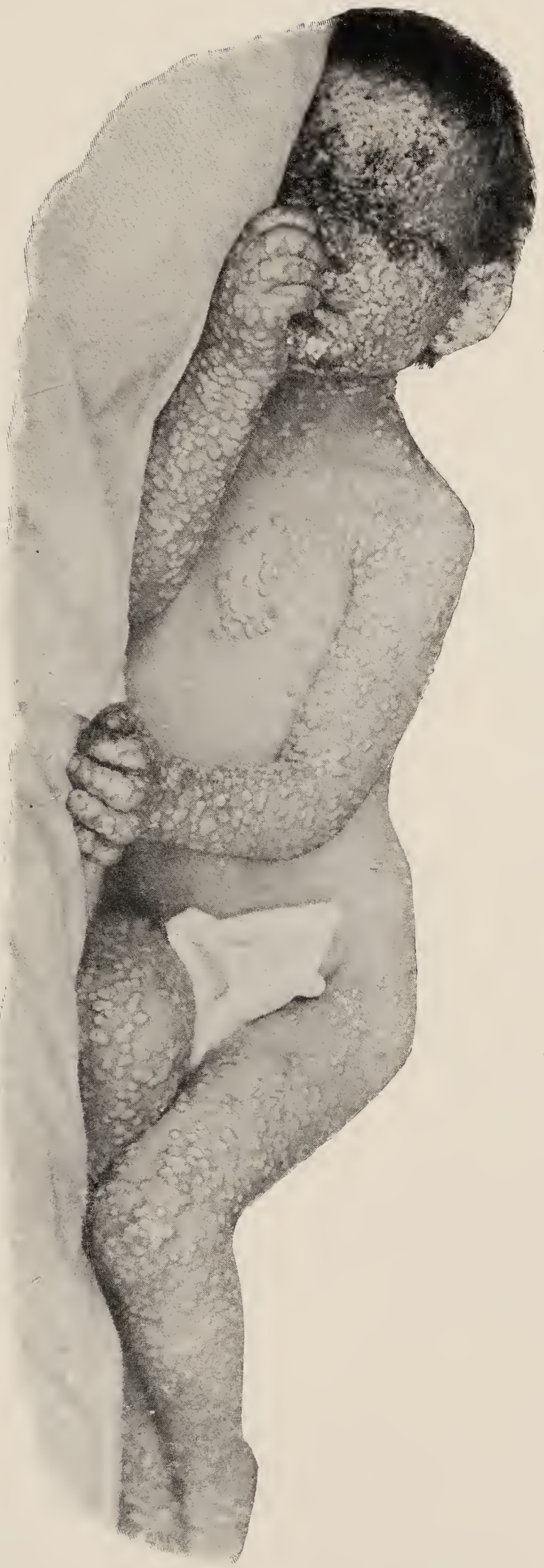
FIG. 281.



Temperature in smallpox. Adult: mild case. (Original.)

Variola, or smallpox, is a specific, infectious, and contagious fever, beginning abruptly with chill, high temperature, headache, vomiting, sweating, and intense pain in the back. On the second or third day of the disease a characteristic shot-like, papular eruption appears, the papules rapidly developing first into vesicles and then into pustules; with the appearance of the rash the temperature falls, but rises again toward the end of the week in the pustular stage (fever of maturation or suppuration). The contents of the pustules are discharged, crusts form and are cast off about the eighteenth day. The disease may be accompanied by a

PLATE XIV.



Variola on the Seventh Day of Eruption. Confluent on Face
and Semi-confluent on Extremities.

number of complications, particularly hemorrhages into the skin (purpuric smallpox) and from the mucous membranes (hemorrhagic smallpox), both forms being popularly called "black smallpox." For convenience of description the disease may be divided into four stages: (1) incubation, (2) invasion, (3) eruption, (4) desquamation.

Incubation. This stage lasts from ten to fourteen days, and is usually unaccompanied by any symptoms except, toward its close, by malaise.

Invasion. The invasion is abrupt, and is marked by chilliness or a distinct rigor, headache, severe pain in the lumbar regions, and sometimes delirium or convulsions, especially in children. The most prominent symptoms are the excruciating headache and backache. The temperature usually rises rapidly to 104° F. or higher in the first twenty-four or forty-eight hours. (See Fig. 281.) Headache and backache continue; there are pain in the epigastrium, a coated tongue, loss of appetite, nausea or vomiting, constipation, and copious perspiration. Prostration is extreme. Erythematous eruptions are not uncommon, especially on the inner surfaces of the legs and thighs. Petechiæ are found in Simon's triangle, the base of which is at the umbilicus and apex at the knees.

FIG. 282.



Discrete variola on the sixth day of eruption. (WELCH.)

The stage of *invasion* lasts generally three days; but it may be shortened to two in very severe cases or lengthened to four in very mild ones, and in complicated and hemorrhagic cases it merges into the stage of eruption. (See Plate XIV.)

Eruption. The characteristic eruption of smallpox appears first as minute specks resembling flea-bites. These in two or three days develop into small papules which feel like shot under the skin. In a day or two

more the papules become vesicles, at first containing a clear fluid, which, however, rapidly becomes turbid; they are umbilicated. In the course of another day or two the vesicles have become pustules and are globular in shape. The period of ripening or maturation, when pustulation is at its height, lasts about three days; it is characterized by a marked secondary fever, the temperature rising as high as, or higher than, in the onset of the disease. The pustules now begin to dry up (desiccation) and form dry scales or scabs, which are cast off toward the end of the third week of the disease (eighteenth day); when the pustules have been sufficiently deep to involve the true skin, characteristic scars, called pits, are left.

The *eruption* appears on the forehead, along the margin of the hair, and in the scalp, then over the rest of the face, especially about the nose and lips, subsequently progressing over the rest of the body from above downward. The eruption is most abundant upon the face and hands, often being confluent here when discrete elsewhere. The face may appear horribly swollen, bloated, and disfigured, and both face and hands are extremely painful from the great distention and the pustules, which are really small dermal abscesses.

Varieties. Three varieties of variola, depending upon the number and disposition of the pocks and upon the presence of complications, are recognized: (1) discrete; (2) confluent; (3) malignant.

In **discrete** variola the pocks are not numerous, and are separated from each other by intervening healthy skin.

In **confluent** smallpox the pustules are close-set, occupy almost the whole body, and coalesce, so that the face looks as though covered with a black, rough mask; the mucous membranes are also covered. The symptoms of the invasion are intensified, and the eruption may appear before the third day. Patients are liable to suffer with profuse salivation, uncontrollable vomiting or diarrhoea (especially children), and with delirium, which is often violent and destructive. The face is dreadfully swollen and the eyelids may slough; the feet and limbs also may be swollen and painful. There may also be severe bronchitis and pneumonia, abscesses, extensive sloughing, and a pyæmic condition.

Malignant, or "black smallpox," is a form in which the blood is so altered that hemorrhages into the skin or from the mucous membranes occur. In the former case there are petechiæ and ecchymoses upon the skin; in the latter more or less profuse hemorrhages occur from the womb, kidney, bowels, lungs, and stomach. The mind of the patient remains clear and he is conscious of his peril. The eruption is delayed or does not occur at all.

Varioloid is a mild form of smallpox occurring in a person partially protected by vaccination, or in a person who, from other causes, does not possess the average susceptibility. It is characterized, apart from its mildness, by great irregularity in the development of the symptoms. The initial symptoms, as a rule, are as severe as in ordinary smallpox. Prodromal eruptions, especially the erythematous, are very common. The eruption may appear first on the face, or on the chest and trunk first, and later upon the face. The fever subsides with its appearance. The

eruption passes from the papular to the vesicular stage, as in ordinary smallpox ; but here the process, as a rule, ceases, the vesicle drying up on the fifth or sixth day of the eruption. If pustules form, they do not reach their full development. The eruption is always discrete. There is usually no secondary fever.

Diagnosis. *H. D.* Exposure ; epidemic ; vaccinia ; or previous attack.

S. D. Backache ; headache ; vomiting.

O. D. Temperature course ; eruption.

P. D. Negative.

L. D. Negative.

D. D. Pneumonia ; typhus fever ; measles ; varicella ; syphilis.

When fully developed, smallpox will not be mistaken for any other disorder. In the initial stage, however, there may be doubt whether the disease will prove to be pneumonia, cerebrospinal meningitis, or typhus. If the patient has been exposed to smallpox and is unprotected by vaccination, and he is suddenly seized with a chill, high temperature, and excruciating pain in the lumbar region, there is great probability in favor of smallpox. If the patient has complained of headache, pains in the ankles and other joints, and is seized with a severe rigor, explosive vomiting, and great weakness of the limbs, the chances favor meningitis in the absence of known exposure to smallpox.

In *pneumonia*, vomiting, chill, and high temperature succeed each other, but excruciating backache is wanting, and, on the other hand, the respiration is increased out of proportion to the pulse, and even in this early stage there may be cough and roughening of the respiratory murmur on one side.

Typhus fever begins abruptly with chill and high temperature ; but the eruption which comes out on the fourth or fifth day is first macular and later petechial, the temperature does not fall with the appearance of the eruption, the aspect of the patient is drunken and stuporous, the conjunctivæ are injected, the eye ferrety, the skin dry, hot, and biting to the touch (*calor mordax*).

Measles. In the papular stage of the eruption smallpox may be mistaken for measles ; but the red, swollen, blear-eyed, photophobic little patient with measles, with the characteristic coryza and obstinate cough, presents a very different appearance from that seen in variola. Moreover, the eruption of measles is relatively flat, smooth, and velvety ; that of smallpox is acuminate, hard, and shot-like. The temperature in smallpox falls as the eruption appears ; that of measles remains high and even increases. The papules of measles do not develop into vesicles.

Chickenpox. In the vesicular stage varioloid may be mistaken for chickenpox. In the latter the eruption is practically vesicular from the start, occurs without prodromata, appears first upon the chest and neck, later upon the face and scalp, is usually very scanty, and rarely becomes umbilicated or pustular. There are, however, severe forms of varicella, in which fever, restlessness, and cough precede the appearance of the rash, which is copious, some of the vesicles being inflamed at the base, some umbilicated, and some with purulent contents. These cases are most common in scrofulous children whose hygienic surroundings are

bad. In such cases the diagnosis can not be made from the eruption. A consideration of the following points must decide: (1) History of exposure to varicella on the one hand or smallpox on the other. (2) The presence or absence of effective vaccination or of scars of antecedent varicella. (3) The age of the patient; smallpox occurs at all ages, varicella only in childhood. (4) The discovery among neighboring children of varicella or varioloid. (5) The rapid evolution of a varicella pock.

Varicella.

Varicella is one of the infections of childhood in which the fever is very mild and may not attract attention. It is an acute, specific, infectious fever, occurring almost exclusively in children, and characterized by the appearance, in successive crops, of colorless or pearly vesicles which dry up and are shed in from two to five days. It is attended with very little constitutional disturbance. A second attack is extremely rare.

Incubation. The incubation is generally about two weeks, but may be one or three weeks. In ordinary cases the first evidence of the invasion of the disease is the appearance of the eruption. In other cases, the severer ones, the child may be noticed for some hours or several days to be indisposed, and complain of loss of appetite, nausea, headache, and vague muscular pains. The *fever* is almost always moderate— 100° to 101° F.

Eruption. The eruption consists first of hyperæmic macules, compared by Trousseau to the rose-rash of typhoid fever. These macules

FIG. 283.



Varicella on the fifth day of eruption. (WELCH.)

rapidly become first papules and then vesicles. The papules are not hard as in variola. They appear at first upon the chest, neck, face, and scalp, then upon the trunk and limbs. The development of the vesicles is so rapid that the eruption appears vesicular from the start. The vesicles vary in size from that of a pinhead to that of a small pea. They are very superficial, and usually rest upon a base that is slightly or not at

all hyperæmic. The contents are at first watery, and subsequently become pearly. The reaction of the fluid is alkaline. Distinct umbilication is rare, and pustulation still more rare, but both occur. The vesicles almost always dry up and form yellowish or brownish scabs, which drop off, leaving a slightly reddened, sometimes depressed spot. Sometimes the vesicles are to be seen upon the buccal mucous membrane and upon the throat. While most of the eruption appears on the first or second day, fresh vesicles continue to appear for several days.

Desiccation. Desiccation usually occurs by the fourth or fifth day, and may be present in the first day or two. As the eruption appears in successive crops, all stages, from the initial macule to the dried scales, can often be seen in one case.

Usually the vesicles are widely scattered, a dozen or two over the entire body. They are most numerous upon the back, and may be as close together as in discrete variola.

In scrofulous and badly nourished children the lesions are more inflammatory and pustules are more common. If they are scratched, ulceration ensues. A gangrenous form has been described by Eustace Smith and others; the cases are apt to be fatal.

In ordinary cases, during the eruption the child is rarely more than indisposed; complications are rare, and the prognosis most excellent. The physician is not often consulted except to have his opinion as to the diagnosis. (For the differential diagnosis from smallpox, see Variola.)

Diagnosis. *H. D.* Exposure; epidemic.

S. D. Negative.

O. D. Eruption; "crops."

P. D. Negative.

D. D. Variola (*q. v.*); vesicular and pustular eczema; impetigo.

Varicella is distinguished from *vesicular* and *pustular eczema* by the fever, the symmetrical grouping and discrete character of the lesions, the comparative absence of itching and burning, and its shorter course.

Impetigo is distinguished by the absence of fever, the more local character of the eruption, and the fact that it is generally pustular. The eruption is more common upon the face and hands than in varicella.

Scarlet Fever.

In this eruptive fever the course of the temperature varies somewhat with the severity of the infection. In many instances *fever* would not be detected without the use of the thermometer. In others it may rise to a greater height, and even be hyperpyretic. Its onset is sudden; it reaches its greatest height when the eruption is complete.

The *temperature* in *scarlet fever* usually conforms to a clearly defined type, increasing gradually until the third or fourth day, when the acme is reached. It declines by lysis in a period of four days. A seven days' chart would be pyramidal in shape. In septic forms (*scarlatina anginosa*), with ulceration of the fauces, the fever continues and becomes remittent. In malignant scarlatina, hyperpyrexia is likely to ensue rapidly.

Scarlet fever is an acute, specific, contagious, and infectious disease,

characterized by a sudden onset, with vomiting, sore throat, and high fever, followed in twelve or twenty-four hours by a bright-red, punctiform eruption, by a very frequent pulse, by a desquamation which is often in large flakes, by a very variable degree of severity, and by a large number of complications and sequelæ, especially nephritis and inflammation of serous membranes.

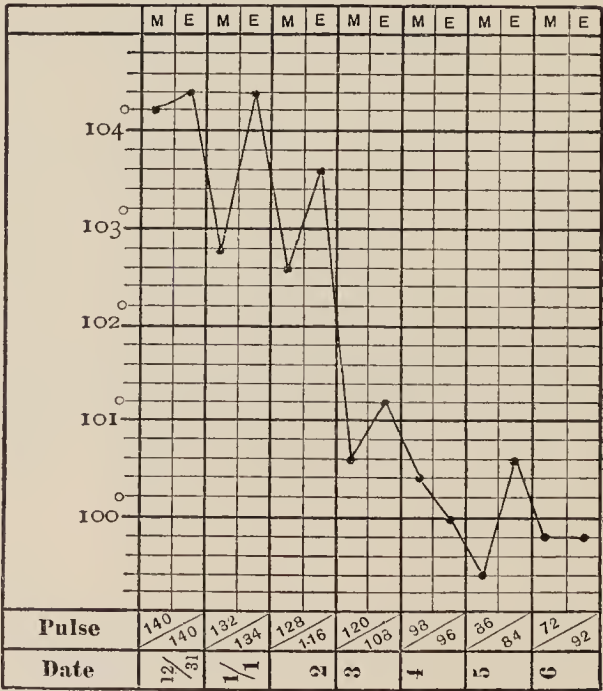
Scarlet fever preferably affects children from one to five years of age. The liability to it diminishes after the tenth year ; but it is very rare under the age of six months. Puerperal women are very susceptible to the poison, and the existence of open wounds favors infection. The disease occurs in epidemics at longer intervals than is true of measles. Cases are most numerous in the autumn and winter months. The peculiar poison is doubtless a living organism, but it has not been isolated as yet. It is very tenacious of life, being capable of infecting, through clothing in which it has been retained, months after the poison has been absorbed by the clothing.

Few diseases vary so greatly in severity in different cases and in different epidemics. It may either be the mildest or the most malignant of diseases.

Incubation. The period of incubation is remarkably short, generally from three to five days ; but it may be only a few hours, and in exceptional cases six days.

Invasion. The invasion is abrupt. It is very common to be told that a child was apparently well on going to bed, but awoke in the middle of the night vomiting profusely and complaining of sore throat. The child is found in the morning with a temperature of 103° or 104° F., a pulse of 120 to 140, and a scarlatinal eruption beginning to show upon the neck and upper part of the chest. Close observation in such cases might have

FIG. 284.



Scarlet fever. Mild attack ; intense eruption. (Original.)

discovered that the child was feverish on going to bed, and somewhat chilly before that. Onset with decided chill, vomiting, and nervous symptoms indicates a severe case.

Symptoms. The *subjective symptoms* of scarlatina are few ; they consist usually of pain in swallowing, with stiffness of the neck muscles, some headache, thirst, malaise, and a moderate amount of weakness. In the eruptive stage the skin itches, burns, and is frequently hyperæsthetic.

The *objective symptoms* and their order of succession are very characteristic. Vomiting is the rule, except in mild cases, and hence is of importance in diagnosis, especially in otherwise doubtful cases. The temperature is high at the onset, frequently 103° or 104° F. It falls a degree or so in the morning ; but the following evening, when the eruption is usually at its height, it rises to 104° or 105° F., and then gradually falls to normal in the course of a week in ordinary cases. (Figs. 77 and 284.)

The *pulse-rate* is characteristically frequent, being 120 to 160 oftener than slower. This frequency is not an indication of danger.

The *blood* shows a leucocytosis, beginning on the first day and continuing through convalescence. A close relationship exists between the degree of leucocytosis and the rash. Suppurative complications tend to increase the number of white cells. The finely granular eosinophiles are greatly increased during the first few days. The mononuclear cells and lymphocytes are diminished at first, but after a short time their percentage increases.

The *throat* exhibits a uniform flush extending over pharynx, tonsils, soft palate, and sometimes forward on the hard palate, nearly to the teeth. Sometimes dark-red points can be distinguished on the soft palate. The tonsils are inflamed and projected toward the median line from each side. Frequently the mouths of the follicles are blocked by a creamy-white exudate. It is not uncommon to find a severe follicular tonsillitis at the first visit.

The *tongue* is at first covered with a thick, creamy fur, through which enlarged red papillæ show. The enlarged papillæ look like small grains of red pepper sprinkled on the tongue. Sometimes the papillæ are elevated and have a button-like appearance. The symptoms appear very early in the disease, and may continue for three or four weeks. The coating soon disappears from the tip, leaving it bright red—the “strawberry tongue.”

The *skin* is hot and dry. The characteristic eruption usually appears within twenty-four hours, often within six to eighteen hours, of the chilliness or vomiting which marks the onset. Sometimes it comes out very slowly, seeming to be just ready to appear, but not appearing in its full development for four or five days.

The intensity of the *eruption* varies from a scarcely perceptible erythema to the color of a boiled lobster, and is proportionate to the severity of the disease. In ordinary cases the patient appears to be covered with a uniform red efflorescence ; but a closer inspection shows that there are darker red spots, between which the skin is more or less erythematous. It is first seen about the ears and neck, and spreads with great rapidity, covering the entire body in a day. It is most intense upon the trunk and flexor surfaces. Upon the extensor surfaces the punctate character is better seen. Pressure causes the redness to disappear, but it immediately reappears. Papular and vesicular forms of eruption are also seen. The

physiognomy in scarlet fever is peculiar. The circle about the eyes, nose, and lips remains pale, and in marked contrast with the rest of the fiery red face. Itching and burning are annoying symptoms at times. The eruption fades gradually, in ordinary cases, when there is no pressure or irritation, disappearing toward the end of the week.

The eruption is succeeded by *desquamation*, which is extensive in proportion to the intensity of the eruption. The flakes are larger than in measles, and in severe cases the epidermis may come off in long strips. This shedding from the hands and feet is sometimes so great that the cutaneous cast is compared to a glove. This stage may be protracted for several weeks, danger of infection lasting as long as desquamation continues.

The *urine* is at first scanty, high-colored, and febrile. Later, when desquamation is in progress, there is great liability to albuminuria as a complication.

Varieties. In addition to the ordinary form already described scarlatina exhibits many irregular forms. There may be only a sore throat or follicular tonsillitis. If a rash is present, it is very faint, and hence easily overlooked. The diagnosis in such cases must be made from the fact of exposure to infection and from the appearance of the throat. The occurrence of vomiting is very important in the diagnosis, as it is rare in ordinary pharyngitis and tonsillitis. Often such cases escape detection altogether, until possibly a dropsy from scarlatinal nephritis indicates their nature.

Severe diarrhœa may prevent the eruption from developing upon the skin. It appears upon the fauces, and the diagnosis is based upon this, the pulse and temperature, and the fact of exposure.

In **scarlatina anginosa** the strength of the poison is spent upon the throat. Pain is great and deglutition difficult. The tonsils are greatly swollen so as almost to occlude the fauces, and their surfaces are covered with creamy exudate. The cervical glands are swollen, and there is a tense and brawny cellulitis. Sometimes the tonsils become gangrenous, and the cervical or submaxillary glands suppurate or become gangrenous, with resulting pyæmia and death. Suppuration may extend to the ears and maxillary sinuses. In this form, also, a false membrane is sometimes found upon the fauces—post-scarlatinal diphtheria. It is probably not due to the Klebs-Löffler bacillus, but to a streptococcus.

In **malignant** forms the attack is ushered in with a chill, followed by hyperpyrexia, convulsions, marked ataxic symptoms, or stupor. The profound blood-disturbance is shown by the dusky hue of the eruption. Some patients lie in coma-vigil, others are very restless and delirious. Vomiting and diarrhœa are sometimes superadded. Patients may emerge from this condition and succumb later to a nephritis or to grave anginose symptoms; but death in a few days is the rule. In rare cases the dose of poison is so enormous that death takes place in a few hours, without the appearance of any eruption.

Complications and Sequelæ. The severe local symptoms mentioned under the anginose variety, together with convulsions, hyperpyrexia, and ataxic symptoms, may properly be regarded as complications. Apart from these the most frequent are nephritis and endocarditis or pericarditis.

Nephritis generally appears with the beginning of desquamation. It is nearly as frequent in mild as in severe cases, probably because the danger of exposure to cold is greater in the former, although the scarlatinal poison unquestionably has a selective affinity for the epithelium of the kidney. The symptoms do not differ from those of acute parenchymatous nephritis occurring under other circumstances. In some cases we have weakness, languor, slight fever, and prolonged convalescence; in others, œdema, anuria, convulsions or coma from uræmia.

Endocarditis is often preceded by tenderness and soreness of the muscles and joints—scarlatinal rheumatism. Endocarditis and pericarditis develop in the course of the fever, giving rise to an increase or continuance of the fever, to local pain or dyspnœa, and to the usual physical signs.

Pleuritis and *meningitis* also may occur. Much more common complications are *otitis*, peripheral *neuritis*, and affections of the joints, grouped as *scarlatinal rheumatism*. Paralyzes, peripheral and central in origin, are occasional sequels of the disease. Scarlatina is found also in association with other diseases.

Diagnosis. *H. D.* Exposure to cause; epidemic.

S. D. Sore throat; vomiting; headache; clinical course.

O. D. Eruption; fauces; tongue.

P. D. Negative.

L. D. Leucocytosis; albumin in urine.

D. D. Muscles; rubella; acute local faucial infections; acute gastritis; acute meningitis; pneumonia; diphtheria.

Sudden onset, rapid rise of temperature, persistent and causeless vomiting, and sore throat lead one to suspect this affection. The characteristic eruption and its mode of evolution, the rapid pulse, the peculiar tongue, the circle of pallor on the face, are characteristic of the eruptive stage. "The appearance of a punctate eruption in the axilla and in the groins, together with the congestion of the tonsils and a punctate eruption in the roof of the mouth, no matter whether there is any eruption anywhere else or not, are positive proofs of scarlet fever." (McCollom.)

Unfortunately, all cases do not develop to the same degree, so that frequently we must wait for the period of desquamation; more infrequently, for the occurrence of sequelæ, as acute nephritis, otitis, or adenitis.

Measles. Scarlet fever is distinguished from measles by the mode of onset, which is sudden, with chilliness, high temperature, vomiting, and sore throat, and great rapidity of the pulse; whereas the onset in measles is gradual, with coryza, cough, moderate fever, perhaps looseness of the bowels, but no sore throat. The eruption of scarlatina occurs on the first day, that of measles on the fourth; the former consists of dark-red spots with intervening erythematous skin, the whole looking at a distance like a uniform bright-red flush; the latter consists of raised, rounded, or flattened spots or blotches, velvety to the touch, and, upon the body and extremities, grouped in patches with crescentic outlines. The temperature in scarlatina subsides gradually after the rash has reached its height; that of measles increases until the eruption is complete, then subsides by crisis.

The rash of scarlet fever persists for six or eight days ; that of measles fades as soon as it is complete, on the fourth day. In the former, desquamation is in flakes or large strips ; in the latter it is branny and nearly invisible. Scarlatina involves by preference the serous membranes and kidneys ; measles, the mucous membranes and lungs.

Scarlatina has to be differentiated from *pharyngitis*, *tonsillitis*, and digestive disturbances, attended with vomiting, high temperature, and, occasionally, erythematous eruptions. In ordinary pharyngitis and tonsillitis the redness is more apt to be confined to the pharynx, tonsils, and arches of the soft palate ; in scarlatina it extends as a flush over the soft and hard palate and buccal surfaces. In the former, high temperature, a very frequent pulse, and vomiting are unusual ; in the latter they are the rule. The glands of the neck also are more apt to be involved in the latter.

In *acute gastritis* there is usually a history pointing to indiscretion in eating, with constipation. The pulse is not so frequent as to suggest scarlatina, sore throat is absent, and any erythema present lacks the characteristic dark-red points, and is not followed by desquamation.

Rubella. The diagnosis from rubella is difficult at times. It differs from scarlatina in presenting mild catarrhal symptoms, sneezing, suffusion of the eyes, and cough, with a relatively fleeting eruption. The latter perhaps appears most frequently upon the back and chest. Often the eruption is the first thing noticed amiss with the child. It more commonly resembles the rash of measles than that of scarlatina, but when it resembles the latter most it is apt to be discrete and of a darker red. There may be a very intense rash without much constitutional disturbance, the temperature being lower and the pulse much slower than would be expected in a scarlatina presenting the same appearance. Nausea may be present, but vomiting is very rare. The post-cervical and post-auricular glands are more commonly enlarged in rubella than in mild scarlatina, though this symptom is not invariable.

Diphtheria is distinguished by its gradual onset, patches of false membrane developing upon the fauces early. In anginose scarlet fever, with severe follicular tonsillitis, the differential diagnosis is essentially the same as between simple follicular tonsillitis and diphtheria (*q. v.*). In addition, the pulse and temperature have a much higher range in scarlatina. The erythema of diphtheria is distinguished from the eruption of scarlatina by its fleeting character and by the absence of desquamation.

Meningitis. Grave cases which begin with repeated vomiting, convulsions, delirium, and insomnia simulate meningitis ; but a satisfactory cause for the latter is lacking, while the excessive heat of the skin, sore throat, very frequent pulse, and early eruption clear up the diagnosis.

Pneumonia. So, also, the onset with vomiting, convulsions, and high temperature resembles pneumonia ; but in the latter the respiration is proportionately more frequent than the pulse, with altered breath-sounds and percussion-sounds, while sore throat and eruption are wanting.

Measles.

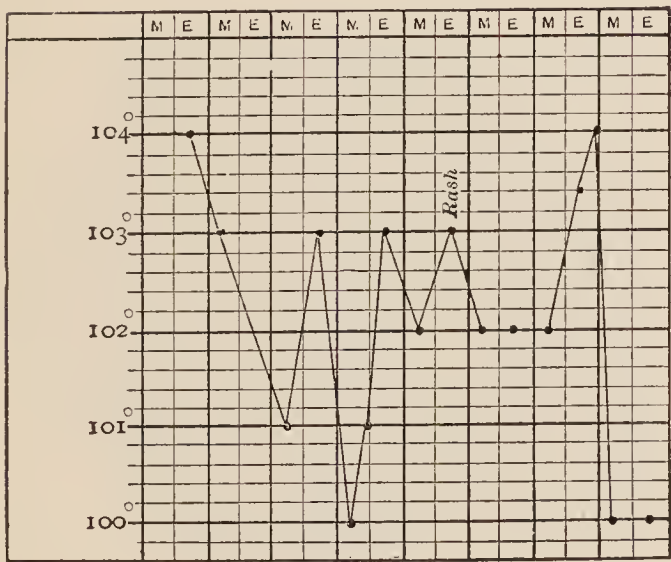
The course of the *fever* in this affection resembles that of smallpox in that after the initial rise of the first twenty-four hours the temperature remains normal until the appearance of the eruption on the third day. It is an acute, specific, infectious, and highly contagious fever, characterized by coryza and bronchitis, a red, papular eruption coming out on the fourth day, and followed by a branny desquamation about the ninth or tenth day. The mucous membranes are especially liable to complications.

Measles occurs in epidemics, especially in cold weather, but individual cases are met with in large cities at all seasons of the year. It is so contagious that when one case develops in a household or institution almost every person exposed to it and not protected by a previous attack acquires it. Children from one to five years of age are most susceptible to the poison, but it may occur *in utero* and in old age; moreover, the same person may have several attacks, showing that one attack does not afford the same protection as an attack of scarlatina or variola.

Measles is sometimes found in association with scarlatina and varicella, but it is especially liable to occur after pertussis.

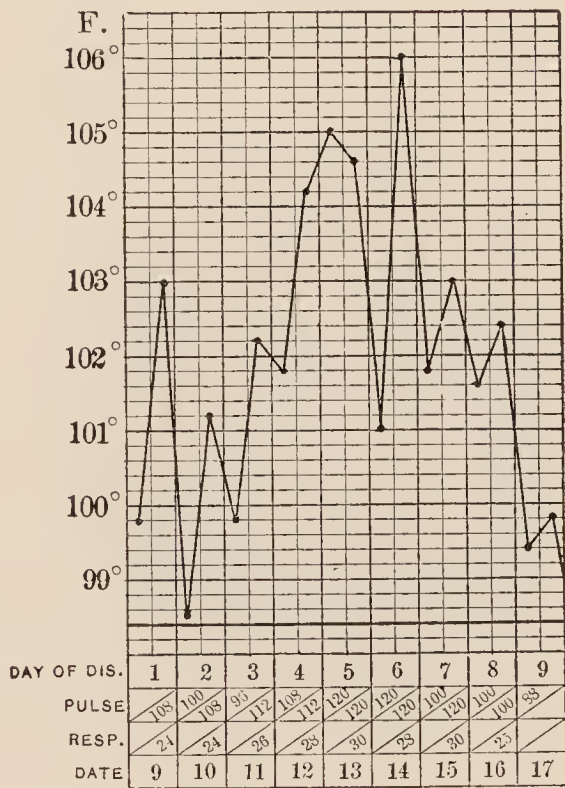
The specific cause of the disease has not yet been isolated.

FIG. 285.



Measles. Temperature taken on the first day, made higher as the result of attendance at school and exertion. (Original.)

FIG. 286.



Measles. Lower temperature second and third days. Hyperpyrexia sixth day. Abundant eruption. Bronchitis severe. (Original.)

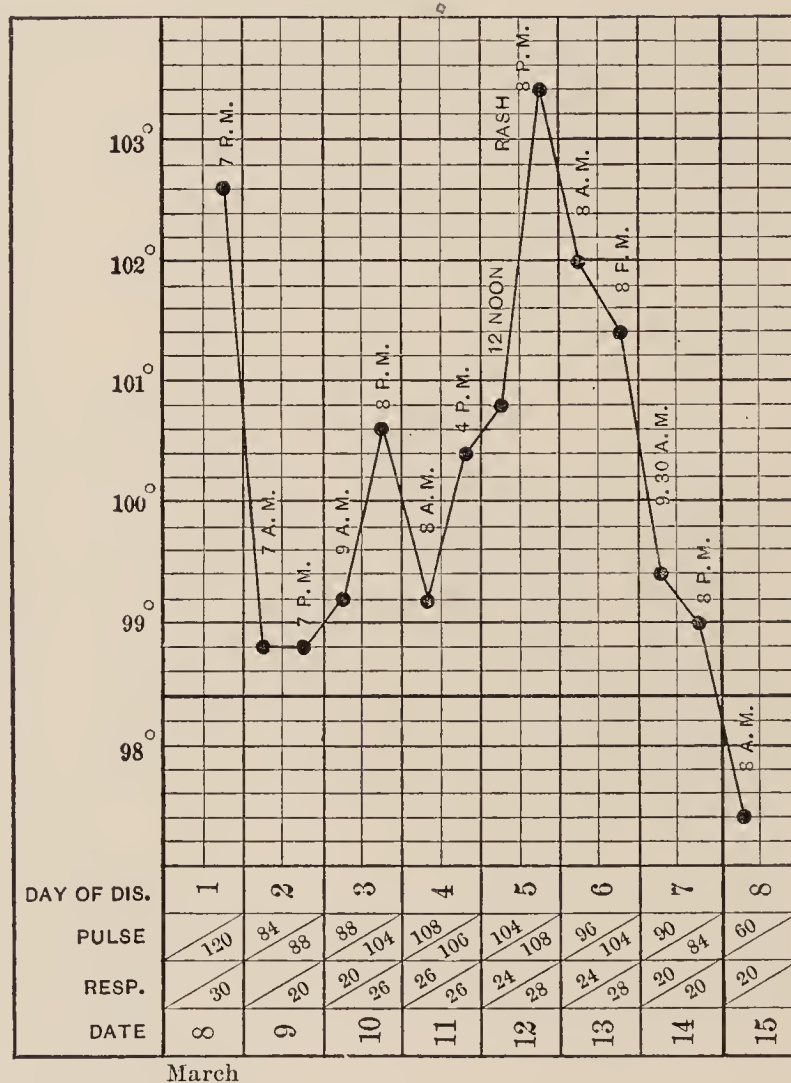
Incubation. The period of incubation lasts from eleven to fourteen days. During this time the patient may exhibit no symptoms, or may be irritable and restless, with disturbed sleep and occasional cough, and looseness of the bowels.

Invasion. The invasion is marked by cough and fever, and by redness of the eyes and lacrymation, sometimes with photophobia, sneezing, and an irritating, watery discharge from the nose, which subsequently becomes mucopurulent, and by cough and fever. In short, the early

symptoms are those of a severe coryza. These symptoms last from three to five days (generally four) before the *eruption* appears.

But an eruption is commonly visible upon the base of the uvula and soft palate, as raised, discrete dark-red papules, several days before it appears upon the body. Another mucous membrane eruption peculiar to this infection has been accurately described by Koplik (1897). His observations have been corroborated, so that "Koplik's sign" is a well-established fact. Its importance can be understood when the necessity for early diagnosis for quarantine purposes is realized. This sign appears twenty-four hours, forty-eight hours, and even three to five days before the skin eruption. It precedes the conjunctivitis and begins at the first rise of temperature. The eruption appears on the mucous membrane of

FIG. 287.



Measles. Characteristic chart. Female, aged twenty-seven. (Original.)

the cheeks and lips. It is not seen on the palate or the fauces. It is at first discrete and then becomes confluent. It is at its height when the skin eruption appears and is spreading. In strong daylight this pathognomonic eruption is seen to consist of small, irregular spots of a bright-red color, in the centre of which is seen a bluish-white speck. The bluish-white speck is very small and delicately colored, requiring direct and strong daylight to see it. A combination of the speck on the rose-red background is a positive sign of the invasion of measles. The spots must not be mistaken for *sprue*, which is opaque, white, coarse, and plaque-like. When the rose-red spots coalesce, Koplik describes the appearance of the mucous membrane to be made up of large areas of rose-

PLATE XV.

Fig. I.



Fig. II.



Fig. III.



Fig. IV.



The Pathognomonic Sign of Measles (Koplik's Spots).

FIG. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

FIG. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

FIG. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

FIG. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

red, studded all over with minute raised bluish-white specks, relieved here and there by the normal hue of the uninvaded mucous membrane. The accompanying figures from Koplik's latest paper illustrates this important sign. (Plate XV.) By this sign measles can be differentiated from rubella, scarlet fever, aphthous stomatitis, forms of erythema and urticaria, drug eruptions, the antitoxin eruption, and syphilides.

The *temperature* rises during the first day to 100° or 102° F., or higher if the case is to be a severe one. The bowels are frequently inclined to be loose and the passages somewhat greenish. The temperature falls on the second day to normal or nearly normal, and then steadily rises until it reaches its acme with the full development of the eruption, when, in uncomplicated cases, it falls rapidly to normal. With the coming out of the eruption the coryza increases in severity, and cough is a prominent and annoying symptom. It consists of a series of five or six explosive efforts without expectoration. In severe cases the cough is almost incessant, so that rest is much interfered with. It depends upon a catarrhal inflammation of the entire respiratory tract, from the nose to the bronchioles.

The Eruption. The eruption on the skin appears first about the neck, face, and wrists, and spreads in two or three days over the entire body. It is usually most copious upon the face, which is swollen, dark red in color, and closely set with papules, which are elevated, rounded at the summits, and feel velvet like to the touch. When to this picture is added that of a severe coryza with mucoserous exudate, which often glues the eyelids together and oozes out upon the face, and a corresponding condition of the nasal orifices, the physiognomy is at once seen to be unusual. At this stage, moreover, photophobia is often considerable, the child burrowing its head in the pillow to escape the light.

The *eruption* is not apt to be confluent upon the body; here the dark-red, elevated, smooth papules are very distinct. Sometimes they are grouped so as to form crescentic outlines. The eruption fades in the order in which it appeared, and is followed by a fine, branny desquamation. With the completion of the eruption the fever falls rapidly to or below normal, the coryza and bronchitis improve correspondingly, and in forty-eight hours convalescence is fully established.

Complications. The complications of measles affect for the most part the mucous membranes of the respiratory and digestive tracts. The bronchitis, which is always present, may become capillary, or be associated with œdema or with areas of catarrhal pneumonia. These are the most frequent and the most dangerous complications. Pneumonia may develop while the eruption is coming out, in which case the eruption is delayed or the spots have a dusky or bluish hue (black measles). More commonly, perhaps, pneumonia is discovered when, the eruption being complete, a crisis should occur. Epistaxis is not usually dangerous. Profuse diarrhœa is very exhausting and delays the evolution of the eruption. Severe conjunctivitis, sometimes with ulceration of the cornea, is not uncommon. Otitis media occurs oftener as a sequel than as a complication. Noma, or cancrum oris, is a rare complication of measles occurring in ill-fed, badly nourished children. It is frequently fatal.

Convulsions may occur as a complication, especially when pneumonia is developing.

Sequelæ. In cases in which there has been diarrhœa, measles is sometimes followed by considerable weakening of the digestive power. The catarrh of the respiratory tract, which almost invariably accompanies it, predisposes to the development of whooping-cough and tuberculosis. Paralysis may follow measles. It may be central or peripheral in origin, but generally is of the hemiplegic type; cases of acute poliomyelitis, acute ascending paralysis, and disseminated myelitis have also been reported.

Varieties. Measles without catarrh is rare. It can not be distinguished from a measles-like rash, seen in rōtheln, except by the occurrence in the neighborhood of other cases of undoubted measles.

Measles without eruption is to be recognized by the coryza, possibly with eruption on the soft palate, the course of the temperature, and the exposure to specific infection.

Black measles is the name given to malignant forms in which, owing to complications, particularly pneumonia, the skin is dusky and the eruption comes out poorly and has a bluish color. In rare instances the eruption shows a hemorrhagic tendency, the spots being livid or ecchymotic. Actual hemorrhages from mucous surfaces may occur, the patient dying in coma or convulsions.

Diagnosis. *H. D.* Epidemic eruption; age.

S. D. Coryza; cough; clinical course.

O. D. Fever range; eruption; Koplik's spots.

P. D. Bronchitis.

L. D. Bronchitis.

D. D. Eruptive fevers.

Rubella.

In a few instances this affection may run its course without *fever*. In the large majority of cases, however, a moderate degree of fever prevails, and in some it may reach a considerable height.

Rubella is an acute, specific, contagious, and infectious fever, characterized by a gradual onset, with moderate fever, sore throat, and slight coryza. The eruption which appears without prodromata, usually resembles measles more than scarlatina. The duration, however, is shorter than measles, the disease milder, and complications are rare.

The disease is amply proved not to be a hybrid of measles and scarlet fever. The incubation-period varies from one to three weeks, but is generally about two. As a rule, this period is passed without symptoms.

The invasion is without prodromata, or none more definite than languor and indisposition, the first thing noticed being the eruption. This in some cases consists of pale-red, smooth, slightly raised blotches, closely resembling measles, but more pronounced on the trunk, and discrete. This is probably a very rare form. More commonly it consists of rose-red macules or papules, occasionally confluent, but usually discrete, and most marked upon the trunk. In still other cases the eruption closely resembles that of scarlatina, differing chiefly in being a paler

red and accompanied by less heat of skin. Sometimes the eruption is circumscribed, as upon the face or limbs. It is usually the seat of considerable itching, and this may be the first symptom that attracts the patient's attention. It will be seen that the eruption is multiform in character. Concurrently with the eruption there is usually slight rise in temperature (100° to 101° F.), suffusion of the eyes, with slight lachrymation and photophobia, and slight pharyngitis; nausea is not uncommon, but vomiting is very rare. Higher temperatures have been recorded in a few cases, and so have nervous symptoms, such as delirium and convulsions, but they are chiefly interesting as very exceptional possibilities.

The eruption extends over the body in twenty-four to thirty-six hours, less rapidly than in scarlatina, and pales much more quickly, fading on the portions of the body first attacked before reaching its height on the last, and being completed in three or four days. Sometimes a branny desquamation succeeds.

In addition to the mild coryza and eruption, the most important objective symptom is swelling of the cervical glands, all of them being sometimes swollen, especially those behind the sternomastoid, the auricle, and along the margin of the hair. This adenopathy, however, can not be relied upon exclusively in the differentiation from scarlatina and measles.

Rubella has few complications: bronchitis, pneumonia, and otitis occur rarely, and still more rarely false membrane on the throat, and albuminuria. The prognosis is excellent. It ends almost invariably in recovery, except in very feeble children.

INFECTIONS RECOGNIZED BY LOCAL SYMPTOMS.

The following infections are characterized by *local* manifestations which are of greater diagnostic significance than the *fever*. These local manifestations must therefore be carefully considered in the diagnosis, as they must be relied upon for recognition of the particular infection. The infections belong to Class I. and Class II. of the classification in Chapter XXXII., Part I.

Mumps.

The swelling is characteristic. It usually begins on one side. The swelling of the parotid gland is observed in front of the ear, then it extends below and around it and behind the ramus of the jaw. Unless there is much collateral œdema the outline of the gland is preserved. The gland is tender and boggy, not indurated. Viewing the face from the front, the midlateral aspects are seen to bulge. The ears stand out from the head. The jaws are fixed. The submaxillary glands are usually enlarged.

The diagnostic features of mumps include the symptoms of the invasion, the general symptoms, and the local signs.

Invasion is sudden, with chilliness, a rise in temperature which is generally moderate (101° to 103° F.), and pain at the angle of the jaw. The corresponding parotid gland as well as the adjacent cellular tissue

begin to swell rapidly. Along with pain on movement of the jaws, any acid liquid, as vinegar, which stimulates salivary secretion, increases the pain. At times the submaxillary glands are involved instead of the parotids, or they may be enlarged and painful several days before the parotid is affected. The disease may be limited to one side or involve the opposite side, as the process in the one first attacked subsides. Rarely it is bilateral from the start. When the swelling has lasted from three to five days, the fever subsides and the swelling begins to disappear rapidly. At this time, however, the opposite side may be attacked or the testicles become inflamed. Usually it is the right testicle. Rarely in girls and women the ovary or mamma is inflamed. Resolution is extremely rapid, and usually the disease is not followed by sequelæ. Sometimes, however, deafness is left. In fact, sudden deafness sometimes announces the commencement of an attack.

If to these attacks we add the data obtained in the social history, the age of the patient (usually under fifteen), and the history of exposure to or the presence of an epidemic, the diagnosis is easily made.

Glandular Fever.

It must not be forgotten that many persons, children especially, have irritable lymphatics (lymphatism) which become enlarged and tender whenever any toxic substance sweeps through the lymph-current. Hence, in milder fever from many causes, as from "cold," catarrh, trauma, or gastro-intestinal disorders, the glands are enlarged. These conditions must not be considered glandular fever, a mistake, I suspect, often made. May not glandular fever be this intoxication?

Glandular fever is an infectious disorder, the cause of which has not been accurately determined. It is characterized by *fever*, usually occurring abruptly, with headache, pains in the limbs and in the lymph-glands of the neck. On examination of the fauces a slight pharyngitis is observed and the tonsils are enlarged. The rise of temperature is accompanied by frequent nausea and vomiting. The temperature rises abruptly to about 102° F. In the second twenty-four hours the glands of the neck, particularly those behind the sternocleidomastoid muscles, enlarge. They are tender. Although there may be some slight œdema, there is no redness or swelling of the skin. The fever continues for three or four days; the glandular enlargement, however, may persist for several weeks, and may end in suppuration.

The infection usually occurs in children between the age of five and eight years, or earlier in life. It may be epidemic. The other lymphatic glands about the neck and in the axilla and groin may be enlarged. In not a few instances there is enlargement of the spleen, and cases of enlarged liver and mesenteric glands have been reported. The absence of an eruption serves to determine the infection from the eruptive fevers associated with adenitis, particularly measles and rubella.

Whooping-cough.

The attention of the physician is called to this infection by the peculiar character of the *respiratory symptoms*. *Fever* is more notable as an expression of one of the complications—bronchopneumonia—than of the general infection. It may, however, be a serious symptom of the infection.

Whooping-cough is a specific catarrhal inflammation of the respiratory passages, involving especially the trachea and bronchi, and characterized by paroxysms of cough, which are succeeded by spasmodic closure of the glottis and a peculiar inspiratory whoop. The disease occurs especially in childhood, is contagious and infectious, and is sometimes epidemic. Whooping-cough may be conveniently divided into three periods :

1. The catarrhal stage.
2. The spasmodic stage.
3. The stage of gradual subsidence of the disease.

First Stage. The patient appears to have an ordinary cold. The amount of redness of the mucous membrane of the eyes, nose, and throat varies considerably, but there is not much discharge from the mucous surfaces. The cough is dry, and sometimes a ringing quality can be detected. The patient is irritable, has slight fever, diminished or capricious appetite, and restless sleep. A mild bronchitis of the larger tubes can be detected by physical exploration.

The cough gradually becomes more frequent and paroxysmal, the eyes are red and suffused, and there is a mucopurulent discharge from the nose. The face often looks slightly swollen, especially about the upper part and under the eyes. Lymphocytic leucocytosis is common.

Second Stage. Transition from the first to the second stage is marked by the appearance of the characteristic whoop. The paroxysmal cough is made up of a series of rapid expiratory efforts, diminishing in force and duration ; when these cease, there succeeds a prolonged crowing inspiration—the whoop. There may be only one paroxysm of coughing at a time, but more commonly, and always in severe cases, one paroxysm is succeeded by another. During the coughing the child's eyes become suffused, the tears overflow, and there is a discharge of serum or mucopus from the nose, and of saliva and bronchial secretion from the mouth. The face becomes swollen and dusky. If the child is walking about, it grasps some object for support during the paroxysm ; or, if old enough, rushes for the water-closet or a basin, because the seizure usually terminates in vomiting. The matters vomited consist of tenacious mucus and the contents of the stomach. The mucus may be streaked with blood, and occasionally there is pure blood. During severe paroxysms, hemorrhages are apt to occur ; these are generally small and most frequently submucous. In well-marked cases, when the disease has lasted some time, the face has a characteristic appearance : it is swollen, sodden, and dusky, with dull, heavy, red, and watery eyes. There is often ulceration of the lingual frænum.

The number of paroxysms varies from two or three to twenty or thirty or more in twenty-four hours, and they are worse at night.

The whoop, while characteristic, is not present in every case, being

absent especially in babies and very young children. Sometimes children have "choking spells" without much coughing and without the whoop. Again, when pneumonia or measles occurs as a complication, the whoop usually ceases for the time, but may reappear later.

Third Stage. The third stage is less well defined than the first two. It may be said to begin when the nocturnal exacerbations become less frequent and severe. The number of paroxysms during the day diminishes, and vomiting is a less frequent accompaniment. Appetite improves, and the child gains in flesh and passes more restful nights.

The duration of the disease is variable. Ordinarily it lasts from six to eight weeks, but it may be prolonged for several months. The patient is liable, whenever he catches a fresh cold, to a temporary return of the spasmodic cough, sometimes with the whoop.

The great majority of the cases occur before the sixth year, and most of these between the second and fourth years.

Rheumatic Fever.

Rheumatic fever is an infection associated with local symptoms of *joint*, *endocardial*, and *pericardial* inflammation. The local symptoms are so intense as to call attention at once to the nature of the infection apart from the course of the *fever*, as it is largely upon these symptoms that the diagnosis is made.

An acute, general, febrile, non-contagious disease, characterized by specific inflammation of the *joints* and contiguous structures, hence called *acute articular rheumatism*. It is further characterized by a tendency of the inflammation to involve the larger joints successively, to skip from one joint to another, and to be associated with endocarditis or pericarditis.

The predisposing causes of rheumatic fever are *heredity*, which is operative in 25 or 30 per cent. of the cases; *age*—81 per cent. of first attacks occur between the eleventh and thirtieth years (Pye-Smith); *sex*—in childhood girls are more frequently affected than boys, but after that period sex appears to have no influence. Multiarticular inflammations, sometimes rheumatic in nature, are met during convalescence from scarlatina and dysentery. They also occur in association with the puerperal state and gonorrhœa, when they are probably pyæmic. The nature of the polyarthrititis which occurs in connection with dengue and hæmophilia is obscure.

Symptoms. The *onset* of the disease is not characterized by constant symptoms. Sometimes the fever and joint-inflammations are preceded a day or two by debility, wandering pains in the joints or muscles, and loss of appetite. Other cases are marked by a chill or repeated attacks of chilliness, followed in a day or two by fever and inflammation of the joints. In rare cases the onset may be followed not by inflammation of the joints, but by inflammation of the serous membranes, particularly those of the heart and its sac.

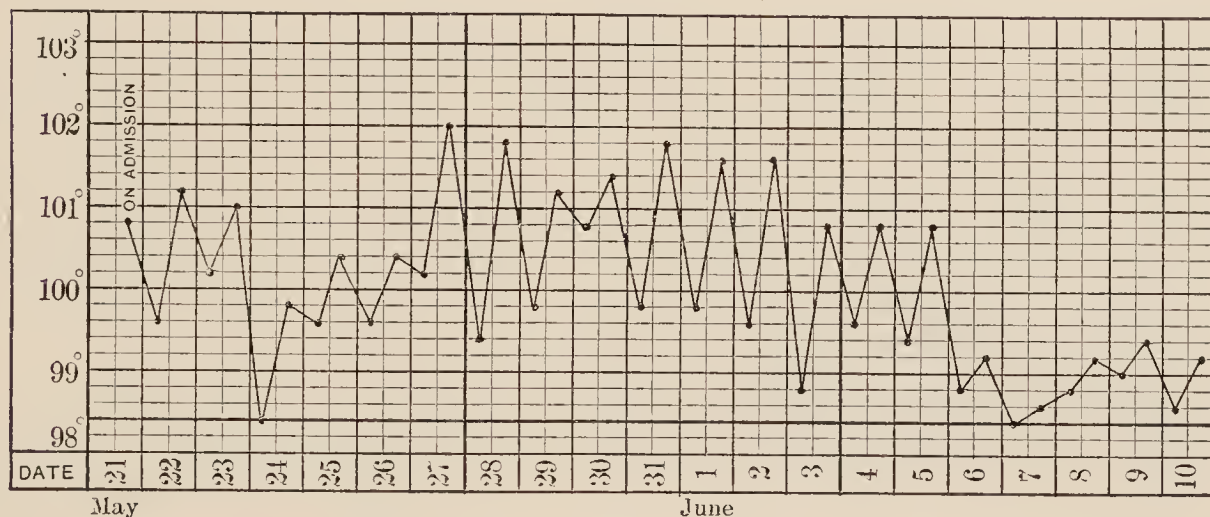
The *temperature* may rise a day or two before there are any joint-symptoms, or fever and arthritis may begin almost simultaneously. The

temperature rises rapidly to 102° , 103° , or 104° F., and one or more of the larger joints, generally the knee and ankle, become painful, tender, swollen, and hot.

The Joint. There may be great pain on motion before there is evident swelling or much local tenderness. The pain varies from mere discomfort to the most excruciating suffering. It is always aggravated by motion or pressure, and is at times so exquisite that the lightest touch, the weight of the bedclothing, or the jar of the bed from a heavy step in the room makes the patient cry out. It may extend beyond the joint to neighboring tendons and nerves. The swelling likewise varies greatly; sometimes there is only slight puffiness with increased distinctness of the cutaneous veins, increased heat in the part, but no general redness; in other cases there is considerable swelling about the joint, so that the bony prominences are obliterated, the surface being tense, red, and very hot to the touch. There is not often effusion into the joint. Swelling is most marked in the wrist and ankle, and less so in the shoulders, hips, elbows, and knees. It is very common on the dorsal surface of the hand.

MULTIPLICITY OF JOINTS AFFECTED. A characteristic peculiarity of rheumatism is its tendency to involve one joint after another. One or several joints may be affected at first; it is very common for the right ankle to be affected, and then in a short time the opposite ankle, followed by the left knee and right knee, and so on with the other joints. The inflammation usually lasts from two to four days in each joint. The

FIG. 288.



Rheumatic fever. Admitted fourth day of disease. (Original.)

process may subside in one articulation and begin in another with startling rapidity. At one visit the physician may find the patient's right ankle swollen, hot, and unbearably painful, and on the next day the right ankle may be quite well and the patient be found suffering acute pain in the right knee or left ankle.

The *pulse* in the early stages of rheumatism is moderately accelerated (99 to 110); it is regular, of good volume, often bounding, and sometimes hard. The *urine* is scanty, high-colored, abnormally acid, and deposits on cooling a copious precipitate of urates, resembling red sand. The *skin* does not feel so hot as one would expect from the temperature. It is continuously covered with a copious, acid, and somewhat pungent

perspiration. *Nervous symptoms* are not marked. There may, however, be slight nocturnal delirium. Sleeplessness from pain is common.

The *temperature* in rheumatic fever is not usually very high; it is much oftener under than over 103° F. In rare cases, however, especially when the fever is complicated with pericarditis, pneumonia, or some disturbance of the heat-regulating apparatus, the temperature may attain the extraordinary range of 106° – 112° F. Such high temperatures may occur suddenly or gradually, and are sometimes attended with marked brain-symptoms (so-called cerebral rheumatism).

Endocarditis and *pericarditis* may occur at any period of rheumatic fever; they may even precede the joint-inflammations. They are most common, however, in the first two weeks of the disease. The younger the patient and the more severe the attack, the greater the liability to heart-complications. They occur in about one-fourth of all cases. Endocarditis is most common; often it is the only lesion, but sometimes it is associated with pericarditis and more rarely with myocarditis. These complications usually give rise to no symptoms at first. Hence, the heart should be examined daily. A sense of constriction in the præcordia or the pit of the stomach, an anxious expression of the face, with pallor, a change in the frequency, but especially in the rhythm of the pulse, and the occurrence of cough or dyspnœa should attract attention to the heart. The physical signs of the respective lesions are fully described under Diseases of the Heart.

The setting in of convalescence from rheumatic fever is marked by an improvement in the tongue, which loses its coating and becomes less red, and by an increase in the secretion of urine, which remains of high specific gravity. The fever subsides gradually, the joints cease to be red, swollen, and tender, the acid sweats lessen, and the appetite improves. In proportion to the duration of the case and its severity the patient is left with debility and marked anæmia, both red cells and hæmoglobin being diminished. In anæmic cases a hæmic murmur may be heard over the base of the heart. In some cases acute dilatation has been observed, with a tricuspid murmur.

Complications and Sequelæ. Apart from heart complications which have been mentioned, *pleuritis*, *pneumonia*, and *bronchitis* occur in from 10 to 15 per cent. of the cases. They are frequently bilateral, and are very much more common in rheumatic fever with pericarditis or endocarditis than in simple rheumatic fever. Moreover, the pulmonary complications are frequently latent, and would be overlooked but for the daily physical examination of the chest. On the other hand, they may develop with great suddenness, and what appeared to be a full-blown pneumonia may subside suddenly as a fresh joint is affected. They behave more like sudden active congestions than true pneumonias. Rheumatic pleurisies are characterized by the rapidity with which effusion takes place, the persistence of pain in the side during effusion, the tendency to involve both sides in succession, the readiness with which the effusion is absorbed, and an acute course.

Nervous System. The most common complication of the nervous system is *delirium*, which is generally associated with insomnia and hyper-

pyrexia, but the latter is not constant. These brain symptoms generally appear in the second week of illness, and about the time of convalescence, or while the joints are still inflamed. The delirium may be low and muttering, accompanied by ataxic symptoms or even by tremors and spasms of muscles; or it may be furious. In favorable cases a deep sleep ushers in recovery; in unfavorable cases the delirium persists with adynamia, the patient dying in collapse or coma, possibly preceded by convulsions.

Chorea sometimes occurs as a complication, but it is more common as a sequel of mild cases in children. *Cerebral meningitis* occurs occasionally, especially when there is ulcerative endocarditis. *Cerebral embolism* is another rare complication.

Various *spinal symptoms* occur in some cases, at times with, and at times without demonstrable lesion of the cord or its membranes. Tetanus, myelitis, and spinal meningitis may all be simulated. Perhaps these symptoms are due to high temperature; but hyperpyrexia is possible without the occurrence of cerebral or spinal symptoms.

Nephritis is rare, but sometimes hemorrhage into the kidney occurs with its usual symptoms. Peritonitis is extremely rare.

Various *erythematous eruptions* are seen from time to time, and occasionally *purpura*. Subcutaneous nodosities have been described by several writers. They are attached to the tendons, fasciæ, and periosteum, and are most frequent on the back of the elbow, the ankles, and patella. They are painless, and may occur in any form of rheumatism.

Diagnosis. Rheumatic fever is distinguished from *gout* by the profuse acid and acrid sweating, the tendency to involve a number of joints, and particularly the larger ones, the greater intensity of constitutional symptoms, the great liability to heart-complications, and the absence of uric acid from the blood.

It is distinguished from *pyæmia* by the wandering character of the inflammation; the acid sweats; the absence of any antecedent condition which would develop purulent foci—such as injuries, abscesses, or specific eruptive fever; the absence of chills, and the fact that in rheumatic fever the sweats are constant, whereas in *pyæmia* they follow a fall in the temperature. Cutaneous abscesses do not occur in rheumatism, and after its subsidence the joint's usefulness is not impaired.

Acute synovitis resembles rheumatic fever, because in both occur symptoms of pain, tenderness, and swelling in connection with a joint. Usually, however, but one joint is involved in synovitis, and there is a history of exposure to cold or injury. The effusion is limited to the synovial sac, is frequently abundant, and fluctuation can easily be detected. The constitutional symptoms are much less marked than in rheumatism. The *acute arthritis of infants*, which is *pyæmic* in character and often of gonococcus origin, must be distinguished from rheumatism.

Milk-leg, or *phlegmasia alba dolens*, differs from rheumatism in that it usually occurs in women after confinement, or as a complication or sequel of fever, as typhoid fever. Usually one leg is affected, or part of the leg, especially the calf. This becomes tense, tender, uniformly swollen, and the seat of great pain. The leg is moved with much difficulty. The

femoral vein may be found to be knotted and tender. There is almost always evidence of antecedent disease.

Acute periostitis when close to a joint simulates rheumatism; but the tenderness and heat are not in the joint itself; they are superficial, and are associated with less swelling. Pitting on pressure is common; and circumscribed fluctuation usually discloses the presence of suppuration. Pyæmic symptoms are added to the local symptoms, particularly if *ostitis* or *osteomyelitis* is present.

The articular symptoms of *glanders* are to be distinguished by the patient's occupation, the mode of onset, the associated symptoms, especially the presence of one or more pustules, and the fact that the painful joints are not so apt to be swollen and red as in rheumatic fever.

In *syphilis* joint-pains frequently occur, but their character is made out by the fact that the joints are not inflamed, and that the pain is much worse, or occurs only at night, by the history, and the therapeutic test.

In some diseases of the brain and spinal cord joint-inflammations of *trophic* origin occur. They are distinguished by the coexistence of some lesion of brain or cord, with hemiplegia or other palsy, and of other trophic changes, such as bed-sores, atrophied muscles, loss of hair, shiny skin, and defective growth of nails.

Subacute Articular Rheumatism. In some instances the joint-inflammation is less severe, and is accompanied by only slight fever. One or more joints may be affected. Subacute rheumatism differs from the ordinary form in being milder in degree and more persistent, lasting sometimes for months. It is generally subacute from the beginning, but may represent a secondary modification of the type in those who have had several attacks of rheumatic fever and have been left in a very sensitive condition.

The type of rheumatic fever is usually subacute in children, and often only one joint is involved. Cardiac complications are more frequent than in adults, and chorea may occur as a sequel. Erythema nodosum and subcutaneous nodosities are more common in children.

Dengue.

The peculiarity of the *fever* in this infection is that it is attended by severe pains in the muscles and joints. It is an acute contagious disease, occurring in epidemics, and characterized by severe pains in the head, back, and joints, by the presence of various skin eruptions, a prolonged convalescence, and a very low rate of mortality.

The disease occurs in epidemics in tropical and subtropical countries, and rarely in cooler climates. It derives its name, dengue (dandy), from the stiff and unnatural gait assumed by convalescent patients. In the southern parts of the United States an expressive name given to the disease is "breakbone fever."

The specific cause of the disease is believed by McLoughlin to be a micrococcus which he isolated. The period of incubation is short, varying from a few minutes to several days, or even a week. Invasion is

sudden and is rarely preceded by prodromata. It is marked by chilliness or a chill, and very severe pains in the head, back, and limbs. In children the onset may be marked by convulsions, which are sometimes followed by stupor and vomiting. The pains are sometimes excruciating, and are accompanied by tenderness of the muscles; there is extreme debility. The temperature rises to 102° or 103° F., but rarely is much higher. The pulse is frequent—110, 120, or more. In from one to three or five days the temperature falls to or below normal (the remission), accompanied by sweating or diarrhoea, and fluctuates about this level for several days, when a second and moderate rise in temperature, which is of short duration, occurs. During the first rise in temperature there is a transient, generally scarlatiniform rash, which is not followed by desquamation. The urine is febrile but not albuminous. During the remission eruptions—scarlatiniform, herpetic, urticarial, or miliaria like—begin to appear, accompanied by the secondary rise in temperature. The eruptions may come out in successive crops, and are followed by desquamation. Convalescence is now established, but may be interrupted by relapses. Strength is regained slowly. The most frequent complications are disorders of the nervous system, but bronchitis and diarrhoea occasionally occur.

Beri-beri.

Beri-beri is an infectious disorder with fever which prevails in epidemic form in tropical and subtropical countries. It is characterized by multiple neuritis associated with anasarca. By most observers it is believed to be an acute infection, although not a few think it is an intoxication due to certain kinds of food; this view prevails in Japan. Conditions predisposing to infections, such as over-crowding, hot and moist weather, and exposure to the elements, are usually present. It is far more common in men, and usually attacks subjects between the ages of sixteen and twenty-five.

Several clinical forms are seen. In the most complete form there is rapid loss of power in the legs and arms, with atrophy of the muscles. The patients complain of pain, and later œdematous symptoms may appear. With the loss of power in the legs there is paræsthesia, with frequent palpitation of the heart and dyspnœa. The pain in the muscles is associated with weakness and tenderness. In milder degrees of this form, pain, weakness in the legs, diminution of the sensibility, and paræsthesia are the most common symptoms. Their onset is gradual and accompanied by catarrhal symptoms. The symptoms may recur from time to time, and are much aggravated during the warm season. The disease may recur in incomplete form for ten or fifteen years.

Following the pain and weakness of the muscles, in some cases œdema becomes very pronounced, associated with effusions into the serous cavities. General anasarca is attended by palpitation and rapid action of the heart and dyspnœa. In this so-called wet or dropsical form atrophy of the muscles is not observed until the œdema disappears. In some instances the infection is very intense, and is characterized by more marked cardiac

symptoms. In these instances acute dilatation may be followed by cardiac paralysis and death in twenty-four or forty-eight hours.

The diagnosis is based upon the occurrence epidemically or endemically in tropical regions of peripheral neuritis with œdema. Thus far no bacteriological diagnosis has been made.

Constitutional Syphilis.

Intermittent, remittent, or continuous *fever* is attendant upon this infection some time during its course. (See Afebrile Infections, Chapter XXXII., Part I.) Failure to recognize the cause of this febrile phenomenon has led to many mistakes in diagnosis.

Constitutional syphilis may be acquired or congenital.

Acquired syphilis is characterized, (1) by the initial lesion, or chancre, which appears usually in about three weeks after contagion; (2) by a period of incubation generally lasting six weeks, but varying from one to three months; (3) by so-called secondary symptoms, comprising febrile symptoms, symmetrical polymorphous skin eruptions, ulcers upon the tonsils, adenitis, less frequently mucous patches in the mouth, or condylomata about the anus, iritis and retinitis, and loss of hair; (4) after an interval varying from several months to twenty years, by so-called tertiary phenomena, which manifest themselves in some cases. These are due to chronic inflammatory indurations of the skin and subcutaneous tissue, resulting in suppuration and ulceration; or of the bones, producing periostitis and necrosis; or of organs, producing gummata and cirrhosis; or of the nervous system, resulting in gummata or chronic degenerative changes. The lesions of this period are unsymmetrical.¹

Course. The course of syphilis in different persons varies as widely as that of any of the eruptive fevers. In some the chancre is a mere papule which heals almost unnoticed; no secondary symptoms appear, and tertiary symptoms also are altogether wanting; or a chronic degeneration of the nervous system develops after the lapse of many years, the patient in the meantime remaining in apparent health. All this may occur, too, without the aid of specific treatment. In other cases the disease is malignant; tertiary symptoms appear very early or appear to take the place of secondary symptoms; ulceration may rapidly melt down and destroy the alæ of the nose or the soft palate; or rebellious periostitis with necrosis may attack the tibiæ, the nasal bones, or the cranium.

Secondary Symptoms. In an ordinary case of acquired syphilis, in about six weeks after the appearance of the chancre the patient complains of languor, weariness, slight fever, pains in the bones, and impaired digestion, and shows a tendency to anæmia. An *eruption* now appears. It is most marked on the trunk and upper extremities, especially the chest and forehead (*corona Veneris*). The eruption may be roseolous, squamous, vesicopapular, papular, pustular, bullous, or tubercular. The color has been aptly compared to that of raw ham. The enlargement of the inguinal, epitrochlear, and postcervical glands, which precedes the eruption, persists. Shallow *ulcers* with a sharply defined grayish outline appear

¹ *Fever* is a constant accompaniment of all forms of syphilis. (See *Fever*.)

on both tonsils. They are painless and do not spread. Ulcers are also liable to appear upon the pharynx, buccal surfaces, tongue, angles of the mouth, penis, vulva, vagina, and around the anus. In the mouth these are apt to be very painful, and may persist for weeks or months in spite of treatment. Relapses are not uncommon. Sometimes there are raised, white patches upon the pharynx. Sometimes the hair becomes very thin and falls out, leaving the patient without eyebrows and more or less bald. *Iritis* and *retinitis* are usually later symptoms. Other symptoms occasionally occurring at this stage are *periostitis*, usually slight, and *onychia*. The most common of the symptoms enumerated are the eruption and the tonsillar ulceration. The eruption comes out gradually during two or three weeks, and persists for about two months. Rarely, however, it is fleeting, or, on the other hand, is unduly prolonged. The secondary symptoms last from six to eighteen months. After their disappearance the patient may remain entirely well for life. In other cases after apparent health, lasting for months or years, the tertiary phenomena already mentioned appear. In the interval the patient may have suffered from various local skin eruptions or ulcers upon the buccal mucous membrane.

The *tertiary lesions* of syphilis are the late *syphilides* (see Skin) and *gummata* of the skin, subcutaneous connective tissue, muscles, or internal organs. Visceral syphilis is seen at this stage. In the brain and spinal cord gummatus tumors, gummatus meningitis, gummatus arteritis, and localized scleroses are found. The symptoms are those of brain tumor when the cerebrum is affected, and of tumor, meningitis, or sclerosis when the cord is affected. In syphilis of the lung we may find gummata scattered through the lung or a fibrous interstitial pneumonia beginning at the root of the lung. Diffuse syphilitic hepatitis or gummata may be found when the liver is affected. The rectum is the most common seat of syphilis of the digestive tract. Myocarditis and localized gummata and endarteritis occur in cardiac syphilis, while in vascular syphilis obliterating endarteritis and gummatus periarteritis are found. Syphilitic orchitis often occurs. Its presence may aid in the diagnosis of obscure visceral syphilis.

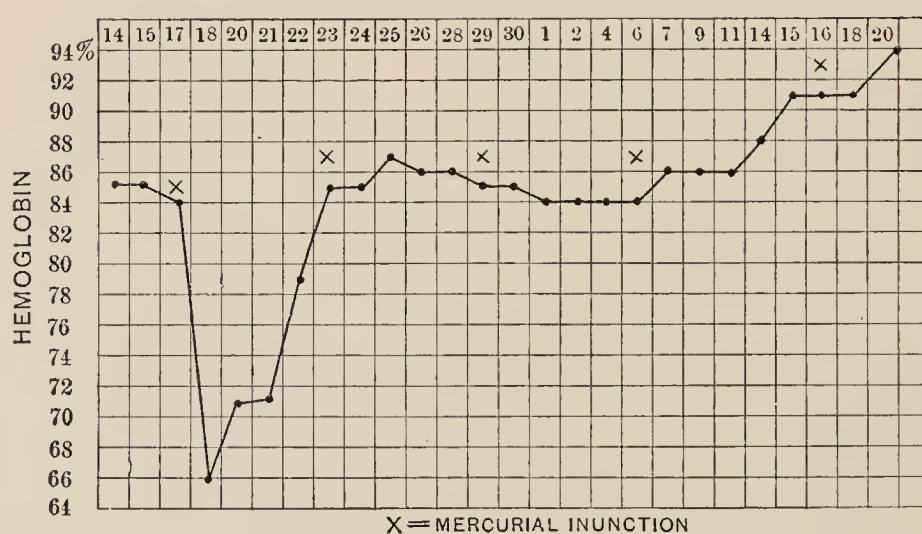
Hereditary syphilis differs in some respects from the acquired form. At birth the syphilitic infant usually exhibits no evidence of its inherited taint. In the course of from one to twelve weeks it develops a catarrhal inflammation of the nasal mucous membrane, which causes snuffling in breathing, and hence is called "snuffles." An eruption soon appears, symmetrical in distribution. It is most frequently erythematous or papular, but it may be squamous, vesicular, pustular, or bullous. In hereditary syphilis the eruption is more apt to be moist and to affect the genitalia and flexures of the thighs than in acquired syphilis; it has, however, the same ham color as is seen in the acquired form. Coincident with the "snuffles" and the eruption appear *stomatitis* and *ulcers* at the angles of the mouth, and sometimes condylomata around the anus. Meantime the child has begun to waste, to be peevish, to be anæmic, and gradually to assume the appearance of a wizened, dried-up old man. As in acquired syphilis, *iritis*, though uncommon, and inflammation of the other structures of the eye may occur, but nodes and disease of the liver are rare.

The infant very frequently dies during this period from exhaustion and inanition.

If the child survives for a year, the secondary symptoms usually disappear and the disease becomes latent. Relapses may occur, and in them, according to Hutchinson, condylomata are likely to appear. The same observer states that the tertiary period may begin at any time after the fifth year, but it is commonly delayed till about the period of puberty. In the meantime the patient may appear fairly well, but usually his development is retarded, there is a tendency to anæmia, and he has often nasopharyngeal catarrh, flattening of the bridge of the nose, premature decay of the upper incisor teeth, and a protuberant forehead. The *teeth* may be perfectly normal in cases which are characteristically syphilitic. The malformation, which was first described by Hutchinson, affects especially the upper central incisors of the permanent set, and "consists in a dwarfing of the tooth, which is usually both narrow and short, and atrophy of its middle lobe. This atrophy leaves a single broad (vertical) notch in the edge of the tooth, and sometimes from this notch a shallow furrow passes upward in both anterior and posterior surfaces nearly to the gum. The notching is usually symmetrical. It may vary much in degree in different cases; sometimes the teeth diverge, and at others they slant toward each other." (See Chapter VIII., Part II.)

Further, the patient may have had or may now be attacked with *keratitis* of both eyes, producing cloudy opacities and accompanied by considerable photophobia. Again, there may be nodes upon the long bones, with nocturnal exacerbations of pain. Cerebral deafness, according to Hutchinson, may occur, but cerebral blindness is rare. There may be ulceration upon the legs, and periostitis and necrosis. The patient usually recovers completely, but he is more liable to die from intercurrent disease than a healthy person, and in general has less resisting power, especially to tuberculosis.

FIG. 289.



Reduction of hæmoglobin after mercurial inunction in syphilis.

Diagnosis. The diagnosis of *hereditary syphilis* is based upon the occurrence of snuffles and skin eruptions, and the existence of keratitis or of cicatrices, especially about the angles of the mouth. A history of repeated miscarriages is suggestive of maternal syphilis. The

diagnosis of *acquired syphilis* is based upon the history of chancre, when that history is obtainable; upon the existence of polymorphous eruptions, or of non-traumatic ulcers upon the legs of young adults, or of scars in the groins or over the tibia, or of nodes, or of alopecia associated with sore throat or mucous patches. The presence of obscure disease of the bones, glands, or spinal cord should lead to the search for a possible syphilitic infection.

Examination of the *blood* during mercurial treatment may, in accordance with Justus' observations, show the presence of syphilis. If this disease is present, the percentage of hæmoglobin falls suddenly and rapidly during the hours immediately following the first administration of the drug. These observations have been confirmed by Cabot. The accompanying chart shows the effect of mercury upon the blood. (See Fig. 289.)

Weil's Disease.

The occurrence of *jaundice* without local hepatic symptoms during the course of *fever* suggests an infectious process. It is a well-known symptom of pyæmia and septicæmia. In the following infection, fever and jaundice are co-ordinate symptoms. *Acute febrile jaundice*, which rapidly becomes malignant, occurring in butchers, laborers, and brewers, has been described by Weil. After exposure to cold generally, as in a beer-vault, the patient is seized with a chill, followed by fever, with headache, vomiting, and epigastric pain. Jaundice sets in rapidly. The temperature remains high or may be intermitting. Stupor, delirium, and coma, albuminuria with suppression of urine, subcutaneous hemorrhages, and hemorrhages from mucous membranes, rapidly ensue. Black vomit occurs early. In one of my cases there was enlargement of the liver, with subcutaneous œdema over the hepatic area. The microscopical appearances were those of acute diffused parenchymatous inflammation. In another, a brewer, the liver was enlarged, but without unusual change, save congestion.

The delirium is sometimes violent. The appearance and symptoms suggest acute yellow atrophy of the liver, but the etiological distinctions are noteworthy: the liver is not small; leucin and tyrosin are not found in the urine; the jaundice is more intense. Yellow fever (*q. v.*) is excluded by the absence of the external conditions attendant upon epidemic and contagious diseases.

Miliary Fever.

The occurrence of *fever* in association with profuse *sweating* is rarely seen without attendant signs of pyogenic infection. When several cases with these symptoms occur at the same time, suggesting an epidemic, the following disease must be considered.

Miliary fever, or *sweating-sickness*, is an infectious disease, occurring in epidemics, and characterized by moderate fever, profuse sweating, tenderness and a sense of oppression in the epigastrium, and a vesicular eruption. The disease has occurred epidemically in England, but is

not met with now outside of France and Italy. After mild prodromal symptoms the disease sets in suddenly with moderate fever, profuse sweating, and epigastric distress, sometimes amounting to anguish. The characteristic eruption appears on the third or fourth day. It consists first of small reddish macules, in the centre of which a vesicle develops. The latter varies in size from that of a pinhead to that of a pea. The contents are at first clear, but subsequently become purulent. Desiccation and desquamation follow. The eruption is generally most profuse upon the neck and trunk. Sometimes there are marked nervous symptoms, and even convulsions and fatal collapse. Miliary fever is distinguished from rheumatism by the moderate fever and absence of joint-swellings, and from malarial fever by the absence of chills, of periodicity in the febrile movement, and absence of malarial organisms from the blood. The duration of the disease is from one to four weeks. The mortality in some epidemics has been very high, in others very low.

INFECTIONS RECOGNIZED BY THEIR RELATION TO ANIMALS.

Milk-sickness.

This is an acute disease affecting cattle, and transmitted from them to human beings in milk or meat. The disease is limited to a few sparsely settled localities west of the Allegheny Mountains. It is characterized by great *debility*, with muscular *tremor* upon motion (hence the name “trembles”), *vomiting* (hence called “puking fever”), a peculiar fetor of the breath, obstinate *constipation*, and moderate *fever* or subnormal temperature. The vomited matters are said to be of a peculiar soapy material, of yellowish or greenish color. The duration is usually less than a week. The patient may sink into a typhoid condition and die in coma, or he may die in a few hours. Convalescence is protracted.

Foot-and-mouth Disease.

A specific, infectious disease, communicated to man through cattle, sheep, or pigs, and characterized by a *stomatitis*. It is communicable by milk; the period of incubation is from three to five days. Invasion is characterized by slight *fever*, heat and soreness of the mouth, and the development of vesicles which burst and leave shallow ulcers. Saliva is freely poured out. The tongue swells greatly, and eating is painful. Vesicles sometimes appear about the fingers, but not upon the feet. The disease lasts from one to two weeks, and ends almost invariably in recovery.

Hydrophobia.

An acute specific disease communicated to human beings by the bites of animals similarly affected. The animals most frequently affected are the dog, fox, wolf, cat, and skunk; 90 per cent. of the cases in human beings are due to dog-bites.

The period of *incubation* is uncommonly long and very variable—from two weeks to two months usually. It is said in some cases to be a year or more. The disease has been divided into three stages—the melancholic, the spasmodic, and the paralytic.

In the *melancholic stage* there is pain, hyperæsthesia, or even reopening of the healed wound. The patient is extremely depressed in spirits and may be irritable. He seems to be laboring under a constant tension of fear, and is keenly sensitive to light, sounds, or draughts. He is affected with thirst, but his attempts to swallow water cause intensely painful spasm of the larynx.

The *second stage* is reached usually on the second day. The laryngeal spasms are increased and lead to intense dyspnœa and to pitiable struggling and gasping on the part of the patient. In addition to the convulsive seizures, the patient foams and froths at the mouth, and his face expresses the extreme terror and mental anguish he feels. The second stage lasts from one to three days, and is followed by the *third stage*, exhaustion intermitting with paroxysms of less severity. The patient may now be able to swallow easily, but there is great weakness of the heart, and death may occur from failure of the heart, from asphyxia, or in a convulsion. The duration, as indicated, is only a few days. The result is practically always fatal, but recovery may be possible. Bites of the face are the most likely to be fatal.

Glanders, actinomycosis, and anthrax are infections, the nature of which is suggested by the relation of the patient to infected animals.

INFECTIONS RECOGNIZED BY EXAMINATION OF THE BLOOD.

Microscopical Examination. *Fresh Blood and Smears.* (See page 564.) The following infections are recognized by the examination of fresh blood: *relapsing fever*, *malaria*, *yellow fever*, and *anthrax*. *Typhoid fever* may also be recognized by this means, but is more frequently diagnosed by means of serum diagnosis and by culture-methods. By *staining* cover-slip preparations of the blood the diagnosis by the direct method is confirmed.

Serum diagnosis enables us to determine the presence of typhoid fever, yellow fever, tropical dysentery, Malta fever, and possibly tuberculosis. (See page 552.)

Bacteriological examination of the blood corroborates the diagnosis of *typhoid fever* made by the above methods. By it we are also enabled to determine the presence of *gonorrhœal infection*, of *cerebrospinal meningitis*, of the *pneumococcus infection*, and, in many instances, of infection due to the *staphylococcus*, *streptococcus*, and *Bacillus coli communis*. The gonococcus infection alone will be considered.

It must be remembered that the micro-organisms can not be found in the blood until late in the course of the disease, and even then the infection must have a certain degree of intensity. Unfortunately, they can not be demonstrated in the majority of cases. Positive cultures for the

above reasons are very valuable. Negative cultures do not exclude septic infections.

Relapsing Fever.

Relapsing fever is the first infection to be considered, because historically it is the most important. It is the first infection in which a micro-organism was found to be causal, and is one to which Koch's laws can be applied. It is an acute infectious and contagious disease, occurring in epidemics, and characterized by the sudden onset of a febrile period lasting five or seven days, which is followed by an intermission lasting usually a week, and this in turn by a relapse lasting three days. Its development is favored by filth and famine, but the specific cause is believed to be the spirillum of Obermeier, which is constantly present in the blood during the febrile stage.

The stage of *incubation* lasts from five to eight days (Pepper), during which the patient may complain of malaise, lassitude, and flying pains. The *invasion* is sudden. It manifests itself by a chill or chills, frontal headache, pains in the back and limbs, vertigo, and great physical weakness. The temperature rises very rapidly, reaching 105° F., 106° F., or even higher, in the first day or two. The face is flushed, epistaxis sometimes occurs, the headache and other pains persist, but delirium is not common. The appetite is usually lost, thirst intense, the tongue coated white but moist, the bowels constipated. A mild catarrhal jaundice is not infrequent. Pepper states that nausea and vomiting are prominent symptoms, the matter vomited at times containing blood. Tenderness with pain in the epigastrium is frequently complained of.

The *urine* is scanty, high-colored, and frequently contains albumin and casts; when jaundice exists, the urine contains bile-pigment and sometimes blood.

There is no peculiar *eruption* in relapsing fever; but in this, as in other fevers, erythemata, petechiæ, and sudamina may be present.

The *pulse* is often very frequent and soft, and hæmic murmurs may be audible.

The *objective symptoms* are few. They consist of a flushed face, sometimes with slight jaundice and epistaxis, tenderness in the epigastrium, with moderate enlargement of the spleen and liver, and considerable cutaneous hyperæsthesia, with tenderness along the nerve-trunks. Bronchitis and sometimes hypostatic congestion of the lungs, with their usual physical signs, may be present.

These symptoms continue without much change until the fifth or seventh day, when a decided *crisis* occurs; the latter, however, is sometimes deferred until the tenth day. The *temperature* within twelve hours falls from 106° or 108° F. to or below normal; the pulse diminishes in frequency from between 120 and 130 to 60 or 70; vertigo, headache, and other pains disappear as by magic. The crisis is marked most frequently by a profuse sweat, sometimes by diarrhœa, epistaxis, metrorrhagia, or intestinal hemorrhage. The patient now enters upon convalescence without fever, and apparently makes rapid strides toward complete recovery. On the seventh day from the crisis, however, a sudden relapse occurs,

with a repetition of the symptoms of the first attack. The temperature may be higher and the febrile symptoms more severe, but the duration is shorter—only three or four days. The spirilla, which disappeared in the apyretic interval, are again found in abundance. A second crisis, with its associated symptoms, now occurs. The spirilla again disappear, and in the majority of the cases there is no further bar to complete recovery. A second, third, and even a seventh relapse may occur, as in a case reported by Pepper. Organic lesions do not follow this disease, unless they have occurred as complications; but even in ordinary cases the patient is left weak, anæmic, and with poor circulation. Relapsing fever occurs at all stages, but is most common in adults.

The duration varies according to the number of paroxysms. If there is only one, it is about eighteen days.

Microscopical Examination of Blood. In the blood at the height of the disease the spirillum of Obermeier is found. It is a slender, wavy, thread-like organism of spiral shape, seven or eight times the length of a red blood-cell, with a very lively forward movement in the direction of the long axis. The length varies from $16\ \mu$ to $40\ \mu$, the width is $0.1\ \mu$. Under a low power the blood may appear to be in motion as the result of their movement. They have so far been found only at the height of the febrile attacks; but von Jaksch states that so long as a relapse is threatened the blood contains peculiar, highly refracting bodies resembling diplococci, which are especially numerous before the attack; in some cases it has seemed to him that these diplococci at the very beginning of an attack develop into short, thick rods, from which the spirilla develop; they may, therefore, prove to be spores. Staining is unnecessary for the detection of the spirilla, but cover-glass preparations of the blood can, if desired, be stained with fuchsin or gentian-violet or Löffler's methylene-blue.

Serum Diagnosis. It sometimes happens that a diagnosis must be made during the afebrile period, when the organisms have disappeared entirely from the peripheral circulation. Löwenthal's method is as follows: A drop of the suspected blood is mixed with one containing the living micro-organisms. The mixture is sealed with wax between slide and cover-glass and left in the thermostat at 37°C . for half an hour. Blood from a patient who has just had a paroxysm will destroy the spirilla, so that they lose their motility and spiral curl, and accumulate in bunches. The reaction is like that of Pfeiffer's phenomenon rather than that of agglutination. It is to be remembered that the bactericidal power of the blood dies out before the next paroxysm.

Typical relapsing fever can also be produced by injecting infected blood into monkeys.

The most frequent *complications* are seen in the lungs, kidneys, and heart. Lobar pneumonia is the most frequent. The heart becomes weakened by the very high fever and thrombosis, or sudden failure results. Embolism is very frequent. Suppurative parotitis, abscess of the spleen, profuse epistaxis, abortion in pregnant women, and neuritis deserve mention.

Under the name "bilious typhoid" a malignant form of relapsing fever has been described. It is characterized by intensity of the symp-

toms of the ordinary form, and by bilious or bloody vomiting, jaundice, and delirium; or by collapse, with a purple nose, a small, frequent, and weak pulse, rigidity of the abdominal muscles, tenderness in the epigastrium, and a cold, clammy skin. In some of the cases described by Graves, intussusception of the intestines was found after death. In other cases uræmia is an active factor.

Diagnosis. Unless the blood be examined, the earlier cases in an epidemic may not be recognized until the occurrence of the characteristic relapse. The diagnosis is based upon the occurrence of an epidemic, the presence of the predisposing factors, the clinical course, and the examination of the blood. Relapsing fever is most likely to be mistaken for *typhus fever*, which occurs under similar conditions. The aspect of the two diseases is very different. In typhus there is a heavy, stupid, sometimes besotted expression, with slight redness of the eyes and a contracted pupil. The patient lies oblivious of his surroundings, with low muttering delirium and ataxic symptoms. In relapsing fever, on the other hand, the sensorium is rarely much disturbed, the spleen and liver are enlarged, and there is hyperæsthesia. Moreover, in typhus there is a spotted eruption, later becoming petechial. In relapsing fever this is absent.

Anthrax.

The next infectious disease, the cause of which can be determined by an examination of the blood, is anthrax. This affection is also of historical importance, and is probably the best worked out of any of the infections common to man and the lower animals. It is also called malignant pustule, charbon, wool-sorters' disease, or splenic fever. It is derived principally from herbivorous animals, and characterized by the development of a pustule or boil, with extensive brawny œdema and subsequent toxæmia; or toxæmia may appear first and metastatic abscesses subsequently. The disease also attacks the gastro-intestinal mucous membrane and the lungs.

Anthrax is caused by the anthrax bacillus and its toxins. Outside of the body it forms endogenous spores, which are extremely tenacious of life, and to which infection is invariably due. They infect not only the carcasses of animals, but also the soil, and utensils used in the care of the animals; and they persist with infective power in the hides, hair, hoofs, and wool. It is possible that it may be transmitted to man by stings of insects, particularly flies and mosquitoes.

The period of incubation varies from a few hours to several days. In the form known as *malignant pustule* the patient has a prickling or burning feeling, which may lead him to think he has been stung by an insect on some exposed part of the body, particularly the hand, face, or neck. At the seat of irritation, first a papule, then a vesicle develops. The vesicle may attain considerable size. The contained fluid quickly passes from clear to bloody, and then escapes leaving a dark-brown or black scab (anthrax).

The original vesicle may be surrounded by a series of smaller ones. Instead of disappearing, the base of the vesicle becomes inflamed and indurated, the induration extending to surrounding tissue and causing a

condition of brawny œdema. A whole arm or one side of the face and neck may be swollen. There may or may not be an associated lymphangitis and adenitis.

The general health does not suffer at first, but in a day or two fever sets in, accompanied by delirium, sweating, great weakness, enlargement of the spleen, severe pains in the limbs, and diarrhœa. Death, preceded by collapse, may occur in from five to eight days (Fagge), or the tissue occupied by the pustule may slough out.

Bollinger and others have called attention to *anthrax œdema*, in which there is no pustule, but only a yellowish or greenish swelling of the tissues. Gangrene may ensue. It is seen most frequently in the eyelids, but may be on the head, hand, or arm.

Intestinal Form. Anthrax of the gastro-intestinal mucous membrane, as described by Bollinger, presents the following symptoms: the patient first complains of malaise, loss of appetite, pains in the limbs, giddiness, and headache. Then vomiting may set in, and a more or less severe diarrhœa, the evacuations often containing blood. There may be pain in the abdomen, which becomes somewhat tumid; the spleen is enlarged. Dyspnœa and lividity appear, with restlessness and excitement or stupor. Epileptiform convulsions may occur, the upper limbs may be affected with tetanic spasms, there may be opisthotonos, and the pupils may be widely dilated. The pyrexia is slight, and death is preceded by extreme collapse. The duration of the disease is usually from two to seven days, but sometimes it is scarcely twenty-four hours.

Wool-sorters' Disease. Still another form of anthrax occurs among the wool-sorters of Bradford, England; it is characterized by intense dyspnœa and a feeling of oppression or constriction. Breathing is labored, but not much accelerated. Only a few coarse râles are to be heard on auscultation. The expectoration may be abundant and bloody, or absent. There is a tendency to collapse, with cold, bluish skin, and a subnormal axillary temperature. The rectal temperature, however, is raised two or three degrees. Death may occur in coma and convulsions, or the patient may die suddenly, the mind being clear. The duration of the disease is from one to five days. Bell says that those who survive for a week generally recover.

FIG. 290.



Bacillus anthracis, highly magnified, to show swellings and concavities at extremities of the single cells. (ABBOTT.)

Examination of Blood. *Bacillus anthracis* is found in the blood of the patient or the pus of the lesions of anthrax or malignant pustule.

Morphology. A bacillus, $2\ \mu$ to $3\ \mu$ up to $20\ \mu$ to $25\ \mu$ in length and $1\ \mu$ to $1\frac{1}{4}\ \mu$ in breadth. The bacilli are often joined end to end in long threads, which are massed in bundles. As found in animals they are short rods with square ends. They stain best with Löffler's blue, but also with the basic anilines and by Gram's method. When in the stage of spore-formation, the threads look like strings of beads.

CULTURES. They support the diagnosis.

Inoculation. When *inoculated*, the organism produces the pustule of anthrax. If inoculated into the abdominal wall of a guinea-pig or rabbit, death follows in forty-eight hours. No reaction is seen at the point of inoculation, but beyond this the tissues are œdematous. Ecchymoses

FIG. 291.

*Bacillus anthracis* in the blood of a guinea-pig. $\times 1040$. (GIBBS.)

are seen, and the underlying muscles are pale. The spleen is enlarged, dark in color, and soft. Cover-slip preparations confirm the diagnosis.

Anthrax bacilli are not so numerous in human blood as in that of the lower animals. They are most likely to be found in the spleen, which is apt to be much swollen.

Diagnosis. In doubtful cases a mouse or guinea-pig should be inoculated with the blood. *Carbuncle* is distinguished by its tendency to develop upon the back or shoulders and other covered portions; anthrax is more apt to occur on uncovered portions of the body. In carbuncle there is a series of openings resembling a sieve, filled with pus and plugs of necrotic tissue. In anthrax there is at first a central black crust. The boggy feeling of carbuncle is different from that of the brawny œdema of anthrax. Finally, in carbuncle, anthrax bacilli are not found in the blood.

The intestinal and thoracic forms are distinguished by the occupation of the patients, the absence of other adequate cause, and the result of the blood-examination, cultures, and inoculation experiments.

Malarial Fevers.

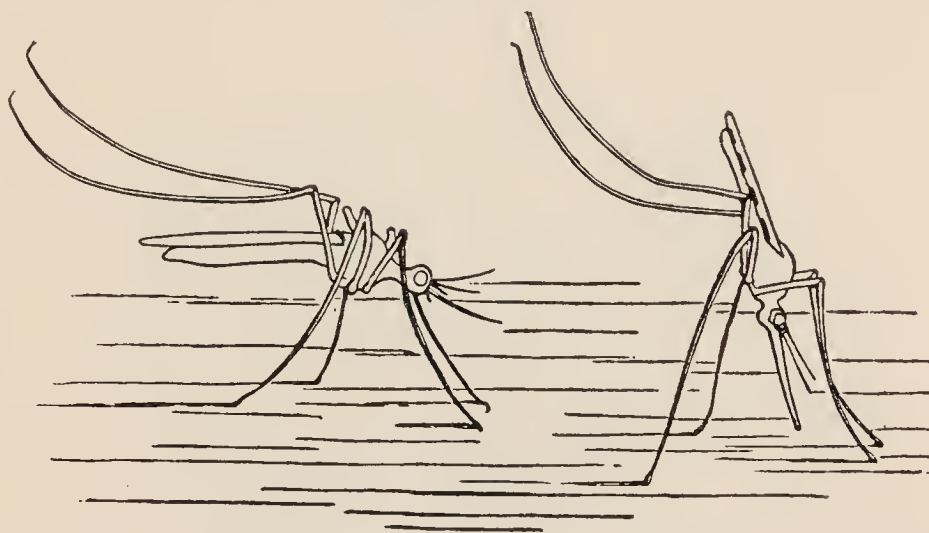
The next infection which will be considered is one of the most common the world over. In its various forms it is recognized by direct

examination of the blood. Its clinical features are such that often but little difficulty surrounds its recognition, but no case should be unqualifiedly pronounced malaria without an examination of the blood. It comprises a group of fevers associated with the protozoan organism of Laveran, and is characterized by periodic paroxysms of chill, fever, and sweat. Malaria is not contagious, but can be transmitted by inoculation.

Malarial fevers, while most prevalent in tropical and subtropical regions, are found also throughout the temperate zone, especially in autumn and spring. In Europe their favorite habitat is Italy, and in the United States the Southern and Southwestern States. Conditions that especially favor their development are marshes and swamps fed partly by sea-water, low ground along streams of slow current, and freshly upturned soil.

The protozoan organism described by Laveran exhibits several different forms, which he regards as stages in the development of one organism, but which may be different species. Golgi maintains that there are several distinct varieties of parasites whose periodicity in development

FIG. 292.



Culex and *Anopheles*. *Anopheles* is recognized by its spotted wings and tilted attitude.

and sporulation corresponds with the different types of fevers. This *Plasmodium malariae* passes through one cycle of its development in the body of a variety of the mosquito known as *Anopheles claviger*. The disease is contracted by the inoculation of the human subject by the infected mosquito.

Intermittent Fever. This is a type of malarial fever in which the temperature remains normal between the paroxysms.

A malarial paroxysm is characterized by (1) chill, (2) fever, and (3) sweating, occurring in the order named and in immediate succession. The time between the beginning of one paroxysm and the beginning of the next is called the "interval," that between the conclusion of a paroxysm and the beginning of the next the "intermission." The interval varies in different forms of intermittent fever: in the quotidian there is a paroxysm every day, with an interval of twenty-four hours; in the tertian there is a paroxysm on alternate days, with an interval of forty-eight hours; in the quartan there is a paroxysm every third day, with an interval of seventy-two hours. In double quotidian there are two paroxysms in the twenty-four hours, but not of the same intensity.

In the double tertian there is a paroxysm every day, the first and third and second and fourth corresponding as to hour and intensity. That is to say, if there be a paroxysm at 10 A. M. Monday there will be another severe paroxysm at 10 A. M. Wednesday, while on Tuesday and Thursday there will be milder paroxysms, but at another hour than 10 A. M.

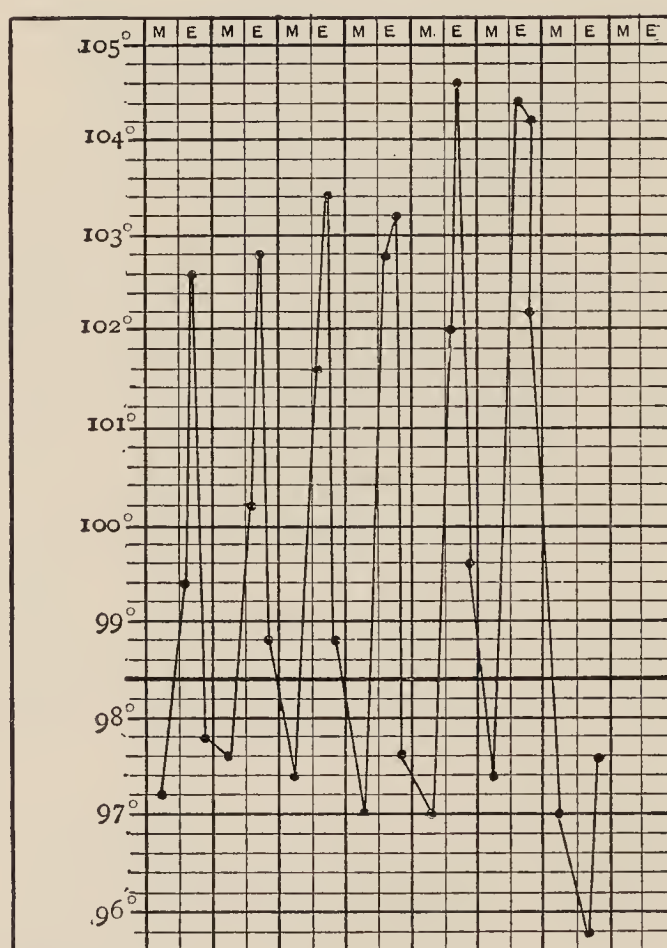
In the double quartan severe and mild paroxysms succeed each other every other day, but each third day is free from paroxysm.

While the rule is for malarial fevers to occur periodically at the same hour, if the disease is growing worse, the second paroxysm may occur an hour or two earlier (anticipation), or, if it is growing better, an hour or two later (postponement). (See Figs. 74, 75, 76.)

Quotidian intermittents are slightly more common than tertian, while the quartan variety is rare.

The *incubation*-period probably varies widely, depending upon the intensity of the poison. As a rule, repeated exposure is necessary to develop the disease in temperate climates. During the period of incubation the patient may suffer from headache, drowsiness, pains and aching in the limbs and back, constipation, a coated tongue, and thirst.

FIG. 293.



Intermittent fever. Temperature every six hours. Morning and evening temperature; highest at chill.

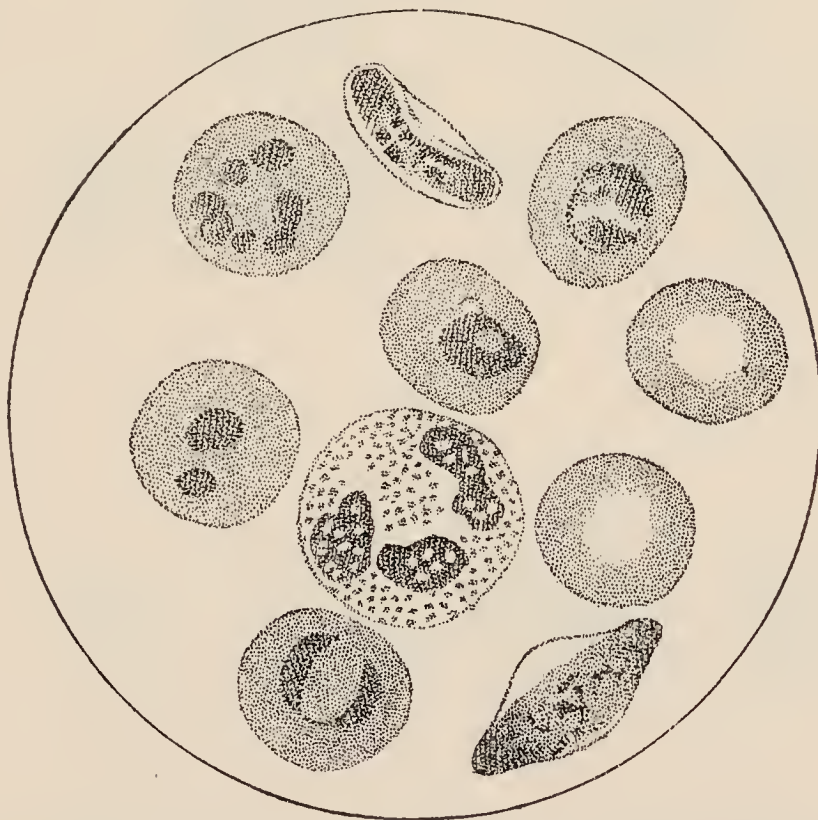
The *onset* of a typical malarial paroxysm is marked by chilly sensations, especially along the spine, accompanied by yawning and the development of "goose-flesh." Then a decided chill sets in, the patient shaking violently. The face is pale and pinched, the lips blue, the nose pointed; as the chill becomes worse the teeth chatter, the whole body feels cold, the skin feeling rough, dry, cold, and harsh. The finger-nails and toe-nails

are blue, the skin being wrinkled upon the palmar and plantar surfaces. The superficial bloodvessels are so contracted that a drop of blood is obtained with difficulty. The voice is thin and weak, almost inaudible.

The volume of blood driven from the surface leads to congestion of the viscera, particularly the spleen, liver, and stomach. Nausea and vomiting are not uncommon. The spleen is perceptibly enlarged, and frequently, also the liver.

Although the surface temperature is depressed, the internal *temperature* is rising, and may be two or three degrees above normal. By degrees the severity of the chill abates and the patient asks to have the extra bed-clothing removed. Reaction sets in; the surface bloodvessels dilate and the skin becomes flushed. The temperature continues to rise, often reaching 103° to 106° , pulse and respiration increasing correspondingly in frequency. The patient complains of a throbbing, dizzy headache, and vomiting may recur. The bowels remain constipated. The temperature now begins to fall, and the sweating stage succeeds. Perspiration appears first upon the forehead, face, and neck, and gradually extends over the rest of the body. The perspiration becomes more and more profuse, until

FIG. 294.

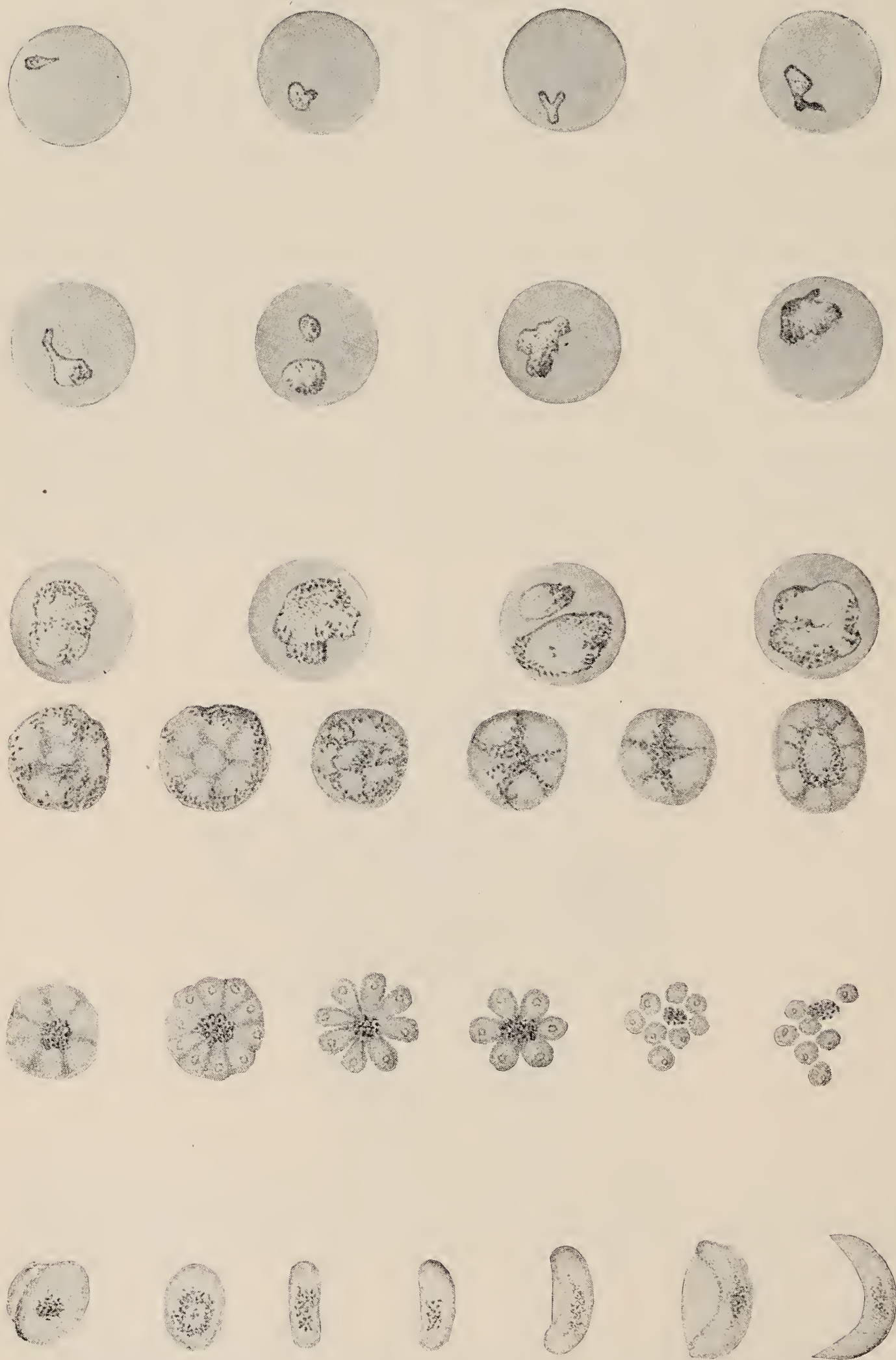


Malarial plasmodia. (Reproduced from colored plate.) To the right, two normal red blood-cells with central depression. In addition, several others with bluish contained bodies and pigment-sprinkled cells, which show the endogenous development of the plasmodia. Besides, two of Laveran's bodies, one exhibiting a delicate little basket appearance. Near the centre a polynuclear white cell with bluish nuclei and red granulation. (H. RIEDER.)

the whole body is drenched with it. All the subjective symptoms vanish with wonderful rapidity, and the patient, with the exception of exhaustion, seems to be restored to complete health. The hot stage lasts from one to two hours, the cold stage from three to eight hours, and the sweating stage from two to six hours.

In the interval between paroxysms the patient is free from fever, but is anæmic, weak, and has impaired appetite and constipation. During the entire paroxysm the mind remains clear.

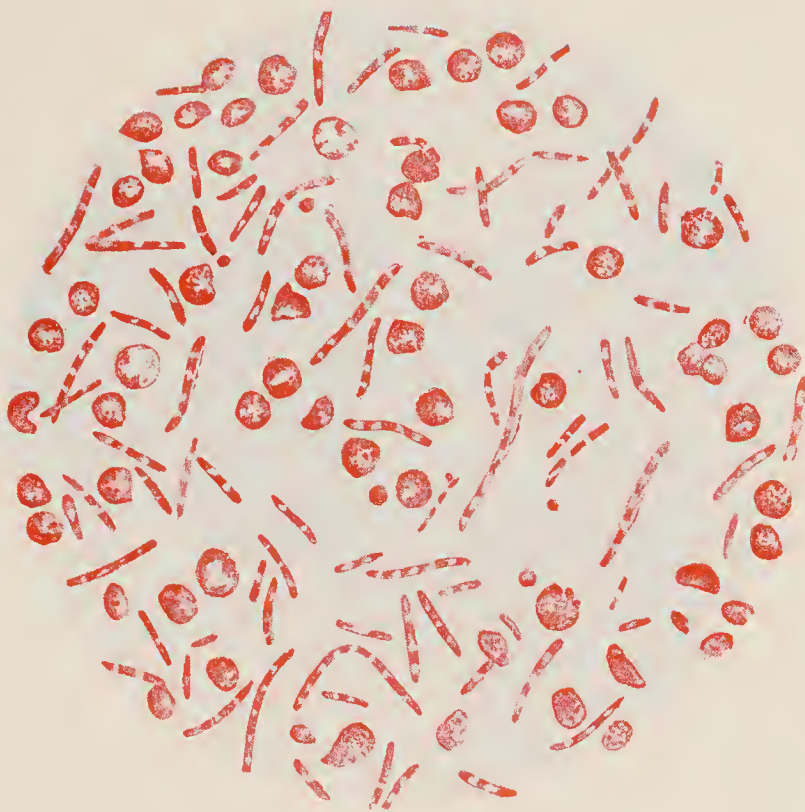
FIG. 295.



The first twelve figures show the malarial plasmodium. It is a pale amœboid body inside the red corpuscle. It increases in size at the expense of the corpuscles. In the last four of the twelve it is enlarged and contains pigment-granules derived from the hæmoglobin. The figures of the fourth row show progressive stages in the process of cleavage of the plasmodium and shifting of the pigment-granules. In the fifth row the process of cleavage is seen to be completed, and final isolation of the spores has taken place. The dark granules are pigment-granules. The last row shows oval parasites—Laveran's corpuscles observed in atypical cases of malaria. (From GOLGI, "Studien über Malaria," *Fortschritte der Medicin*, Bd. iv., Tafel III.)

PLATE XVI.

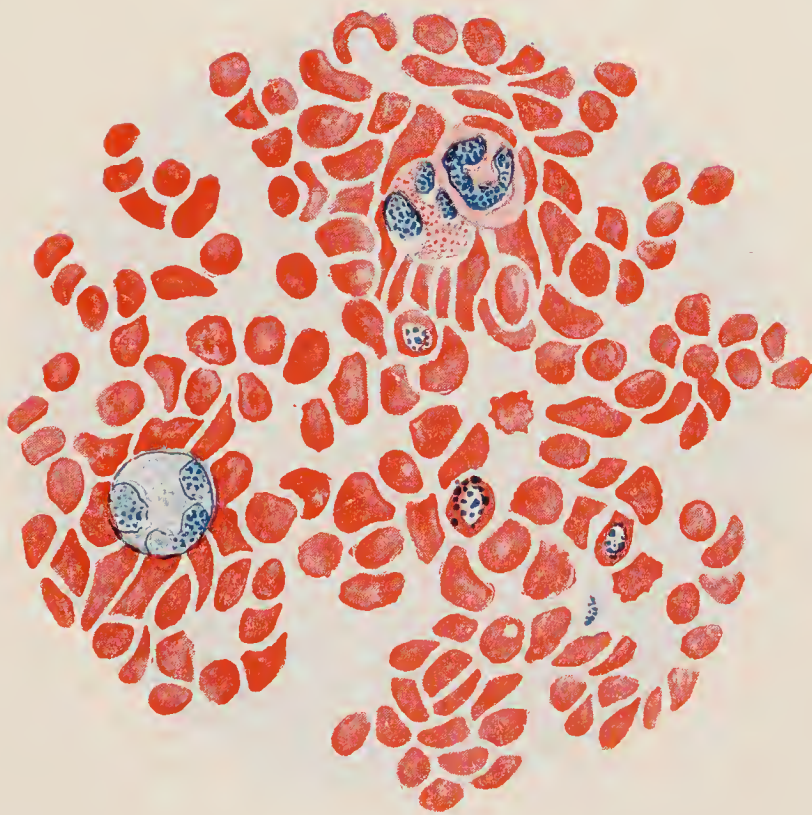
FIG. 1.



Anthrax-bacilli from Rabbit's Spleen.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

FIG. 2.



Protozoa of Malaria, Intracellular and Crescentic Forms.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

The chief *objective symptom*, apart from the phenomena of chill, fever, and sweat already described, is the occurrence of plasmodia in the blood. (See Plate XVI., Fig. 2 ; and Fig. 294.)

Examination of the Blood. The plasmodia of malaria were first pointed out by Laveran. They have been studied in Italy, especially by Marchiafava and Golgi, and in this country by Councilman, Osler, and Dock. Minute amœboid bodies are found in the red corpuscles. These become pigmented with altered hæmoglobin, and grow until they fill nearly the whole of the cell, the pigment being arranged chiefly in a peripheral ring. Later, the amœboid bodies become spherical and transparent, the pigment collecting in the centre. Sporulation now begins, and a fresh crop of small rounded parasites appears, to begin the same cycle over again in fresh corpuscles. (Plate XVI., Fig. 2.)

Three forms of parasites are described : 1. The *tertian*, which sporulate at the end of forty-eight hours, begin as small amœboid intracorpuseular bodies, gradually enlarge, produce fine brownish pigment-granules, and finally completely fill the corpuscle. In sporulation the segments number fifteen to twenty.

2. The *quartan*, which sporulate at intervals of seventy-two hours, are smaller ; amœboid movement is not so marked ; when full grown, the parasites are smaller, and the corpuscles tend to shrink about them and to become a deeper greenish color. They sporulate with five to ten segments, in a very beautiful characteristic rosette appearance.

3. The *æstivo-autumnal* are smaller, and contain less pigment. The period of sporulation is still in dispute. They usually form ovoid, crescentic or round bodies with coarse pigment-granules in the centre.

Golgi maintains that in tertian malarial fever the period between invasion of the corpuscles and the sporulation is two days ; in quartan, three days, the difference in cycle being due to a difference in the parasites.

The onset of the fever corresponds in time to the division of the parasites.

The crescentic form described by Laveran is said to be more common in the irregular forms of malarial fever. Canalis says that it only makes its appearance several days after the beginning of the fever. It is somewhat longer than a red blood-cell, and the pigment tends to collect in a focus about the middle of the parasite. Subsequently it becomes oval and divides into eight or more daughter-cells.

Another form with flagella is occasionally found. Councilman says it is most common in blood drawn directly from the spleen.

The plasmodium of malaria may be *stained* as follows : Cover-glass preparations of the blood spread very thinly are dried in the air and fixed by immersion for twenty minutes or half an hour in a mixture of equal parts of alcohol and ether. They are then stained for twenty to thirty minutes in concentrated aqueous solution of methylene-blue, 60 parts ; 0.5 per cent. solution of eosin in 75 per cent. alcohol, 20 parts ; distilled water, 40 parts ; 20 per cent. solution of potassium hydroxide, 12 drops. The cover-glasses are then washed in water, dried, and are then ready for mounting. The red blood-cells are stained rose, the nuclei of leucocytes a deep dark-blue, and any plasmodia a delicate sky-blue.

Aronson and Phillips' staining method is as follows: Make concentrated aqueous solutions of orange-G, acid rubin, and crystallized methyl-green, leave them to settle, then mix in these proportions: orange-G, 55; acid rubin, 50; distilled water, 100; and alcohol, 50. To this add methyl-green, 65; distilled water, 50; and alcohol, 12. Leave the mixture standing for a week. A well-diluted solution should be used for staining purposes; one drop of the mixture should be added to 25 c.c. of water; the stain should be left on for twenty-four hours and the fixing of the preparations, done before staining, carried out at a temperature of 120°C . As a result the red corpuscles are stained orange, nuclei greenish blue, neutrophile corpuscles violet, and eosinophile red.

One of the best stains is that of Romanovsky: 1 per cent. watery solution of eosin, 2 parts; saturated aqueous solution of methylene-blue, 1 part, in watch-glass. Put the specimens, prepared by heat or alcohol, face downward, floating on top of the solution; cover with another glass. Keep in a moist chamber. Stain one-half to three hours; better two hours. The plasmodia are stained a clear blue; the corpuscles a pale red.

Another good stain is made as follows: 0.5 per cent. aqueous solution of eosin, 1 part; saturated solution (aqueous) of methylene-blue diluted one-half with distilled water, 1 part. Stain as is Romanovsky's method for twenty-four hours. The results are the same.

Futcher has called attention to an extremely valuable method. Upon a dried cover-glass specimen a little of a 1 per cent. solution of formalin in 90 per cent. alcohol is poured and allowed to remain for one-half to one minute. The specimen is dried between leaves of filter-paper; absolute alcohol is then poured over the glass, which is once more dried, and then stained for twenty to thirty seconds in Marchand's solution of phenol-thionin, which is prepared as follows: saturated solution of thionin in 50 per cent. alcohol, 20 parts; 2 per cent. solution of carbolic acid, 100 parts. The solution must stand several days.

The specimen is then washed in water, dried between filter-paper, and mounted in balsam. If the specimen has not been stained too long, the corpuscles take a very slight greenish hue, while the parasites are of a deep-violet color. The preparation should not be stained too long, as in pigmented parasites granules of pigment may be obscured by the depth of the color. This method is particularly valuable inasmuch as it brings out with great clearness the small ring-shaped hyaline bodies of *æstivo-autumnal* fever with their chromatin dot.

Examination of the blood discloses the presence of a high degree of anæmia. The hæmoglobin is usually diminished in greater proportion than the corpuscles. There is a marked reduction in the leucocytes. This leucopenia is most marked after a paroxysm. There are a relative diminution of the polynuclear forms and a relative increase in the mononuclear forms. In severe post-malarial anæmias, as Thayer points out, the blood is characteristic of pernicious anæmia.

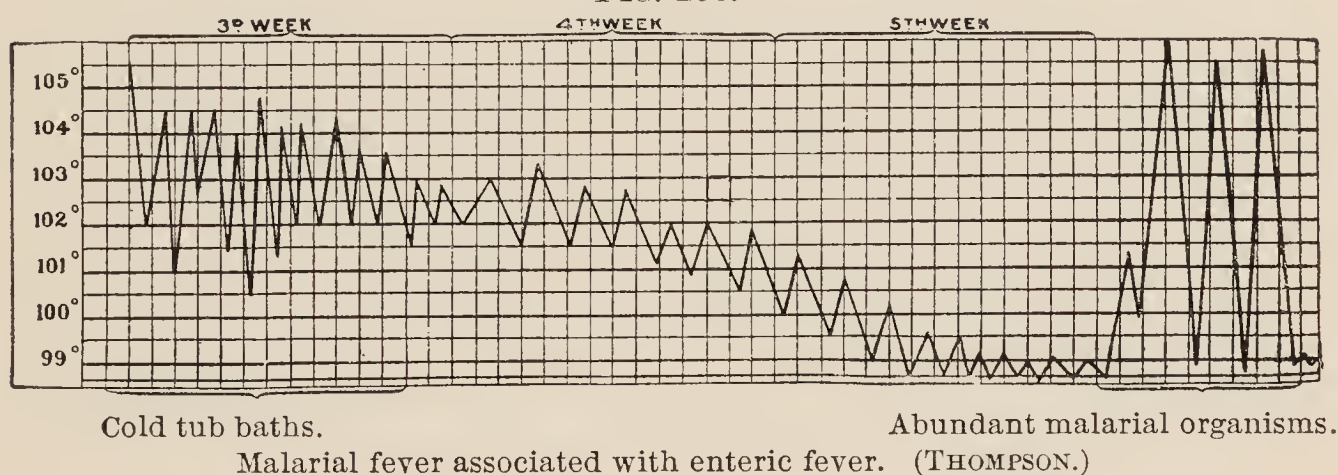
Irregular Form. Irregular forms of intermittent fever are more common in Philadelphia than the typical form just described.

In the *mild form* the patient complains of great lassitude, irritability of temper, and drowsiness during the day, but at night tosses upon his

bed and gets up in the morning more tired than when he went to bed. The back and limbs ache, and the latter feel as though they would give way under him. There is severe throbbing headache, with some dizziness and faintness. The bowels are constipated; the tongue heavily coated with yellow fur. The temperature is moderately elevated and the patient has great thirst. Nausea and vomiting are absent, though there is little desire for food. There may be a burning feeling referred to the splenic region. The patient is worse on alternate days, and the attacks may be preceded by slight creeping chills. On inquiry the patient will be found to live in a low-lying district near one of the rivers, or in a damp house over an unclean, moist cellar, or adjoining a place where soil has been freshly upturned.

In the form known as "*dumb ague*," there is a periodically great depression, with aching in the head and limbs, a sensation of coldness rather than chilliness, but no marked fever and sweating. Nausea and vomiting may, however, be present. Da Costa says he has seen it manifest itself by excruciating pain over the kidney and almost entire suppression of urine. There may also be severe paroxysms of gastralgia. It is more common in old residents of malarial districts.

FIG. 296.

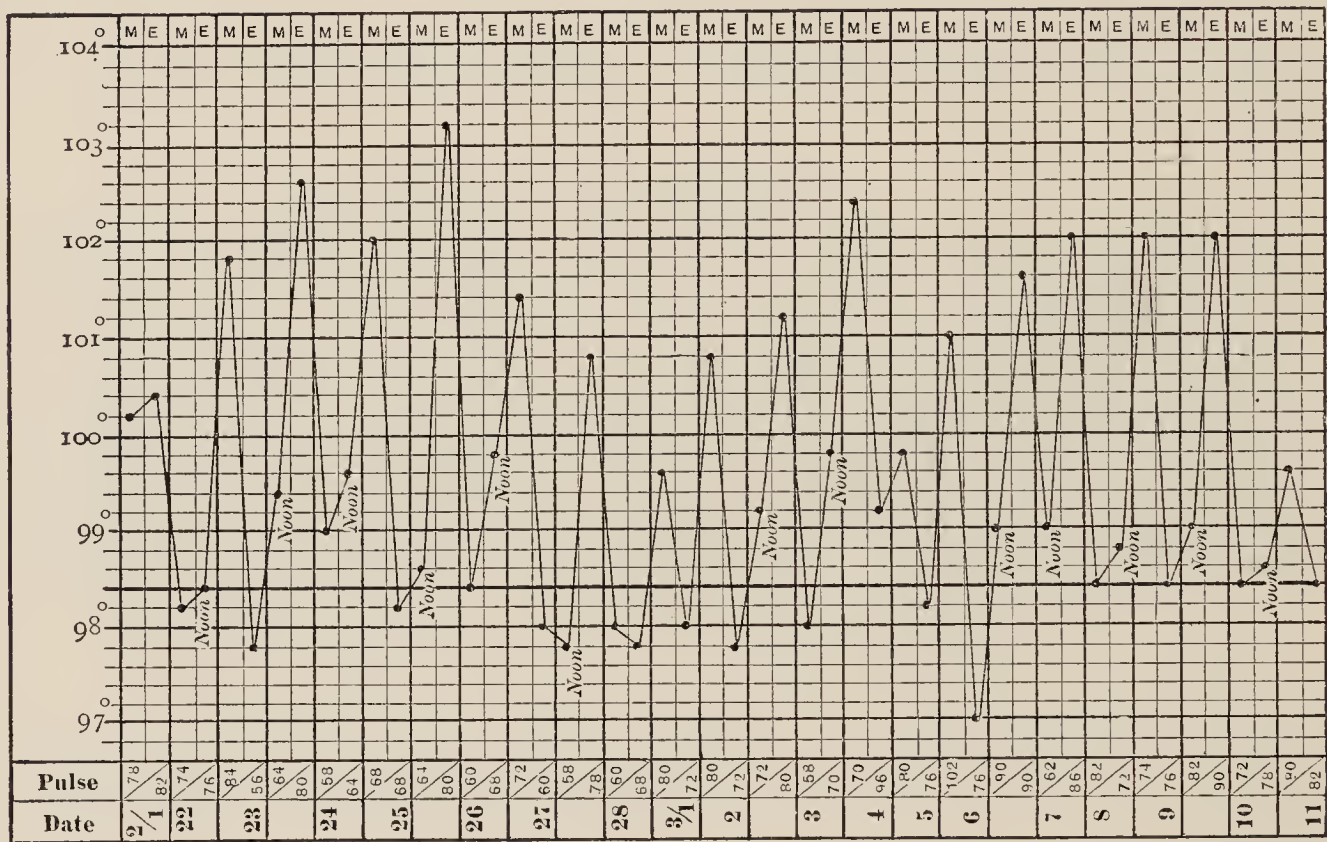


In *masked* malarial fever the poison manifests itself in an attack of neuralgia, especially of the supraorbital nerve and gastric nerves. Malaria may also be latent until some impairment of the resisting power brings it to light. Hence, it appears as a complication of pneumonia and dysentery and typhoid fever (Fig. 296), especially in the southern and southwestern portions of the United States. Moreover, women who have previously had intermittent fever may suffer a recurrence after confinement; it must not be confounded with so-called *puerperal malarial fever*, which is unfortunately more common and of infectious origin.

Diagnosis. The essential points in the diagnosis of intermittent fever are the periodical recurrence of paroxysms of chill, fever, and sweating, or of attacks of dumb ague, or of paroxysms of neuralgia, without organic lesion, associated with the presence in the blood of pigment and plasmodia, and with enlargement of the spleen and possibly of the liver. The so-called therapeutic diagnosis may be made—an intermittent fever which does not yield to proper doses of quinine in three days is not malarial. A typical malarial intermittent fever is not likely to be mistaken for anything else. (See Fever, pages 263, 264.) It needs, how-

ever, to be distinguished from *septicæmic fever*, due to absorption into the blood of pus and the toxins produced by bacteriological growth. Such fever occurs in *tuberculosis*, especially in the stage when cavities form and pus collects; in the puerperal state, in empyema, subphrenic abscess, abscess of the liver, or, indeed, in any form of suppuration. Here, also, there are recurring chills, with fever and sweating, but the attacks are not regularly periodical and intermittent; sometimes the fever is intermittent and sometimes remittent, the chills recur at irregular intervals, and are not so violent as in the malarial attack. The essential difference, however, lies in the fact that a local cause can be found to explain them, such as tuberculosis either of the lung or of some other viscus, or a collection of pus in an organ or cavity, or a fetid discharge from the womb, with local tenderness or peritonitis; moreover, the patient loses flesh more or less rapidly, his blood is free from malarial germs and pigment, and quinine does not control the fever. (Plate XVI., Fig. 2.)

FIG. 297.



A form of intermittent fever from syphilis. J. D., aged twenty-six years. Secondary period. Mercury and potassium iodide relieved it. Observe that the pulse-frequency is not increased.

It is more difficult to make a diagnosis between malaria and the intermittent fever of *hepatic* origin (described elsewhere by the author) because physical signs of any local trouble may be wanting. But the fever of hepatic origin is not regularly intermittent, is not controlled by quinine, but may be by measures directed to the origin of the trouble, and jaundice may be present.

Urethral fever, occurring as the result of operations upon the urethra, or simply from the passage of a catheter or bougie, may be mistaken for malarial fever; but the paroxysm is usually single, and the history of the operation and the absence of plasmodia from the blood clear up the diagnosis.

Syphilitic fever is distinguished by a tendency for the chill, fever, and

sweating to be nocturnal in recurrence, and by evidence of a syphilitic infection coupled with absence of malarial organisms from the blood.

Remittent Malarial Fever—Æstivo-autumnal Type. A type of malarial fever characterized by a remission instead of an intermission in the febrile paroxysms. It is due either to a great intensity of the malarial poison or to a different species of organism. It is much more rare in temperate climates than either quotidian or tertian intermittent, and is attended with more gastric disturbance and a much larger mortality (twelve times greater, according to the statistics of the War of the Rebellion).

The *onset* is more abrupt than in intermittent fever. Prodromata are not so common, but when they occur they are of the same character. The chill is not usually so violent, nor the cold stage so long as in intermittent fever; on the other hand, nausea and vomiting are common, and in some cases there are bilious vomiting and diarrhœa, tenderness over the stomach and spleen, and sometimes jaundice. The temperature rises rapidly from 103° to 106° F., and remains high for a longer time than in intermittent fever, the hot stage lasting in severe cases from six to eighteen or twenty hours. During this stage the patient suffers from headache, pains in the back and limbs, great thirst, and gastric irritability. A remission now succeeds. The temperature falls two or three degrees, but not to normal; free sweating occurs, the nausea and vomiting cease, and the patient becomes much more comfortable. He may fall asleep from exhaustion, but if awake is conscious of weakness, aching in the limbs, and perhaps nausea. In the course of some hours the temperature again rises, often to a higher point than before, but frequently without antecedent chill. The same subjective symptoms are repeated, and another remission follows. Daily paroxysms usually occur, those on alternate days being severe. The temperature often reaches its highest point at the third paroxysm. The disease generally runs its course in from nine to twelve days, but it may last much longer. The type of fever may change to intermittent, which is a favorable sign, or become continued and again remittent, or remain remittent throughout; finally, the fever may subside gradually, or, less commonly, by crisis. The urine is febrile but not albuminous. (See Examination of Blood, page 743.)

Pernicious Malarial Fever. This, as the name implies, is a form of malarial fever with destructive tendency. It is also called malignant and congestive fever. It may be intermittent or remittent. Nearly 24 per cent. of the cases occurring in the U. S. Army from May 1, 1860, to June 20, 1866, proved fatal.

Bemiss¹ divides this variety of malaria into three classes: the *algid*, or congestive, form; (2) the *comatose* form; (3) the *hemorrhagic* form. To this another class, (4) the *gastro-enteric* form, may be added. It is important to remark that the first paroxysm does not usually, in any of these forms, indicate that the type of the disease is pernicious. The first seizure may, however, prove fatal.

1. The *algid form*, according to Bemiss, occurs more frequently than any other, its perniciousness being due to an aggravation of the cold stage of an intermittent attack. The patient is extremely weak, with cold

¹ Pepper's System of Medicine, 1885, vol. i., p. 666.

extremities, pinched features, blue lips, and faint voice. Respiration is shallow, the pulse rather slow, feeble, and irregular; he is further exhausted by vomiting and liquid, offensive diarrhœa, the passages sometimes being involuntary. There may be copious perspiration, but the internal temperature is very high. The mind may be clear, or there may be deep stupor. Unless speedy relief can be afforded the attack ends fatally.

2. In the *comatose form* the patient is completely unconscious, the skin hot "and of a muddy, semi-jaundiced hue." (Bemiss.) Both pulse and temperature are increased. In other cases coma is preceded by wild delirium, resembling acute meningitis. The comatose form is most apt to occur in those who continue to reside in a malarious region without proper safeguards against its poisonous influences.

3. In the *hemorrhagic form* there has been, as a rule, previous alteration of the blood, the bloodvessels, and other tissues, from long-continued malarial poisoning or cachexia. Then, when intense congestion of these parts occurs as the result of the surface-chill, hemorrhage follows. In some districts, however, and at certain seasons, there has been a special predilection of the poison for the kidney, with resulting hæmaturia. The prominent symptoms are a prolonged chill with high temperature; nausea and vomiting, sometimes with the expulsion of a greenish-black fluid; œdema of the lower extremities; general anasarca, occasionally œdema of the lungs, and hydrothorax; bloody and albuminous urine, with tube-casts; and intense jaundice. Pain in the right hypochondrium or over the kidneys is common. Bemiss asserts that uncomplicated malarial fever has not a hemorrhagic tendency.

4. The *gastro-enteric form* has for its prominent symptoms nausea, vomiting, diarrhœa, intense thirst, extreme restlessness, a frequent feeble pulse, and urgent dyspnœa. "The breathing is deep-drawn; each expiration is followed by two short inspirations." (Da Costa.) The patient is cold and partly collapsed. Reaction may or may not occur. The patient may have several paroxysms of pernicious malarial fever and succumb in any one of them. Convalescence is slow. The most frequent sequelæ of malarial fevers are anæmia, neuritis, paralyses, and malarial cachexia.

Typhoid fever is distinguished from pernicious malarial fever by its gradual onset, the absence of chills and vomiting, as a rule; and, on the other hand, the presence of epistaxis, delirium, and ataxic symptoms, tympanites and diarrhœa, with pale-yellow, watery stools, and rose-colored spots. The temperature in typhoid is more continuously high, the daily oscillations being of shorter range. A history of exposure to malarial infection and of previous attacks can often be obtained. The urine of typhoid exhibits the diazo reaction; that of malarial fever does not. The results of the blood examination (Widal's test) and the bacteriological studies would settle the diagnosis in most doubtful cases. It must be remembered that a mixed infection occurs sometimes, in consequence of which the plasmodium of malaria may be found either at the beginning or in the decline of the typhoid infection.

Malarial cachexia occurs especially in those who have lived for a long time in malarious regions, and who may or may not have had typical

malarial attacks. The patient suffers from dyspepsia and constipation, with occasional bilious attacks; the face is of a pale lemon-yellow color, and may be slightly jaundiced; there is marked anæmia, with pigment and crescentic and flagellate forms of plasmodia in the blood, together with great enlargement of the spleen (ague-cake) and some enlargement of the liver. The patient is weak and languid, and sometimes has considerable mental depression.

SERUM DIAGNOSIS.

The infections just described are recognized by an examination of fresh blood or cover-slip preparations. The next group of infections may be recognized by serum diagnosis. Too much stress must not be placed upon this method of diagnosis, yet its value is so great that one is fully justified in giving it a high place among the precise methods of diagnosis of infections.

Typhoid Fever.

The first of the infections to which such diagnosis has been applied *in extenso* is typhoid infection or typhoid septicæmia. This infection is caused by *Bacillus typhosus*. Its most common expression is a symptom-complex which attends a septic process and local intestinal ulceration combined. This symptom-complex is known as *typhoid fever*. In rare instances the infection is stated by some to be unattended by fever. More frequently a febrile course, following a definite continued type and lasting from twenty-one to twenty-eight days, prevails. In mild or abortive forms the fever rarely reaches 103° F., and declines from the seventh to the fourteenth day. In the grave forms the fever is often very high and attended by cerebrospinal, renal, pulmonic, or severe gastro-intestinal symptoms.

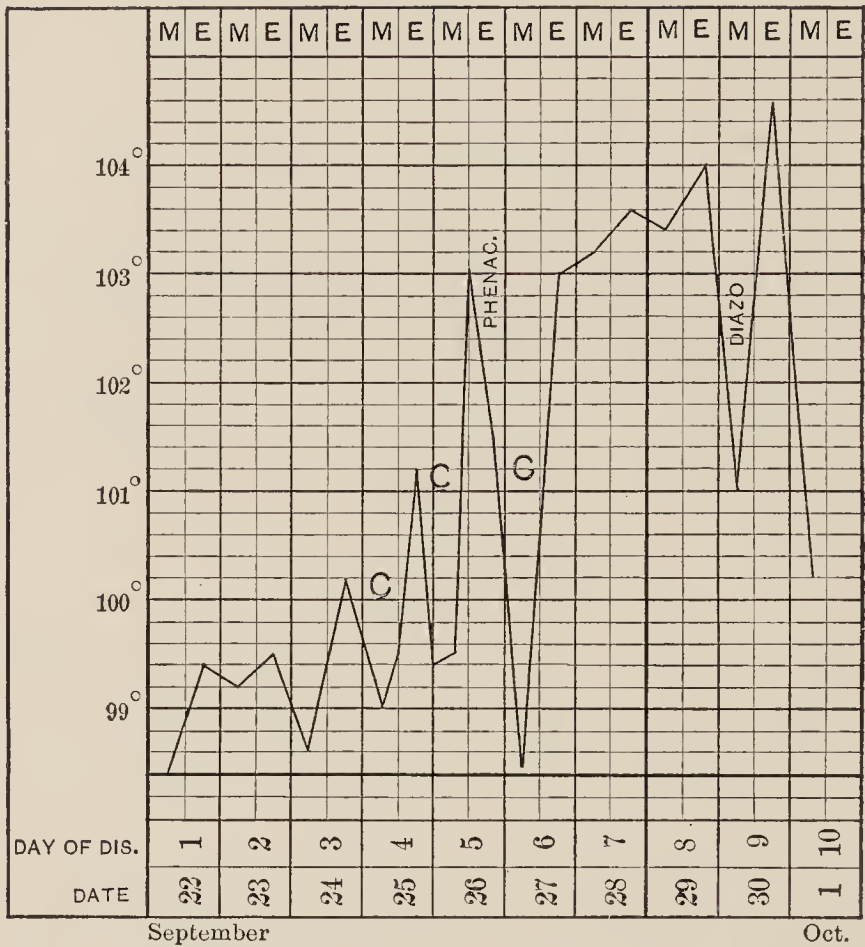
The most important infection prevailing in the temperate zone is the one we are now about to consider. It is an acute, specific, infectious, and mildly contagious disease, characterized by a gradual onset, a continued fever, an eruption of rose-colored spots, marked nervous and abdominal symptoms, and an average duration of three or four weeks.

It occurs sporadically and epidemically, and in large cities is apt to be endemic. Its special habitat is in temperate climates, but it may occur anywhere. It is relatively rare in the southern and southwestern portions of the United States. It is more frequent in the latter part of the summer and in the autumn and winter, and following hot and dry summer weather. Young adults are especially prone to it, but cases have occurred at all ages. Change of residence from the country to the city is a predisposing factor. Those living in cities often acquire immunity, but they may lose it upon moving elsewhere. The state of previous health does not seem to have any influence.

Incubation. The period of incubation in typhoid fever varies from four or five days to three weeks; more commonly it is from one to two weeks. During this time the patient usually is languid, becomes tired easily upon exertion, has severe headache, sleeps poorly, and his rest is

disturbed by dreams. There is often, even thus early, a dull and listless expression of the face. Toward the close of this period, and in severe cases, there may be colicky pain in the abdomen, a tendency to looseness of the bowels. cough, epistaxis, mental sluggishness, and chilliness.

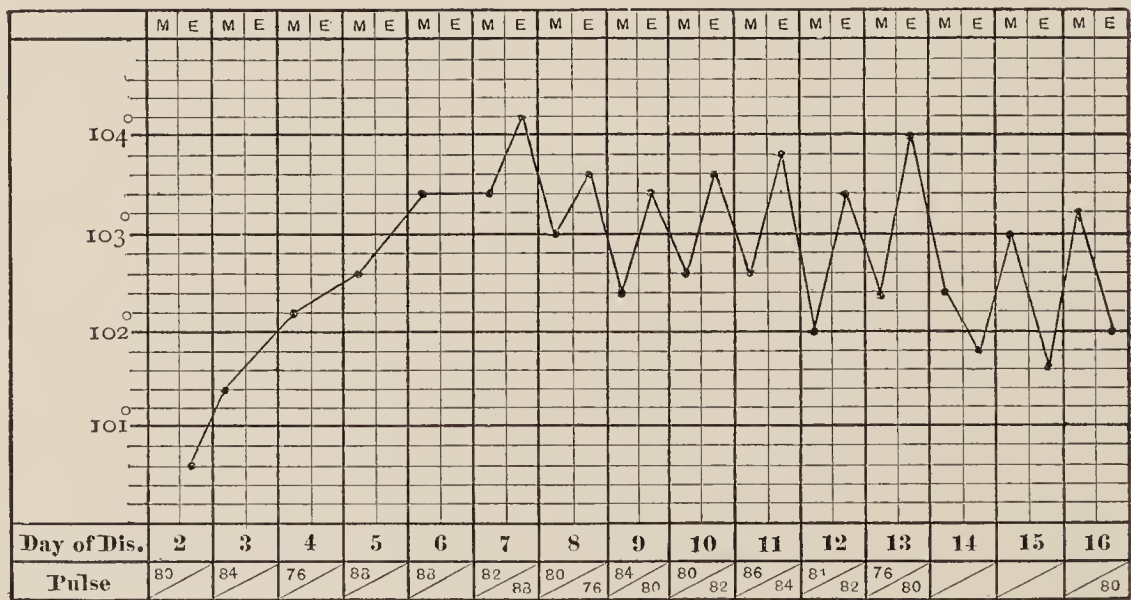
FIG. 298.



Temperature ranges; first week of typhoid fever. (Dock.)

Pepper was led repeatedly to anticipate the approach of typhoid fever by the unusual dulness of hearing and by the persistent occipital headache coming on after a few days of general malaise.

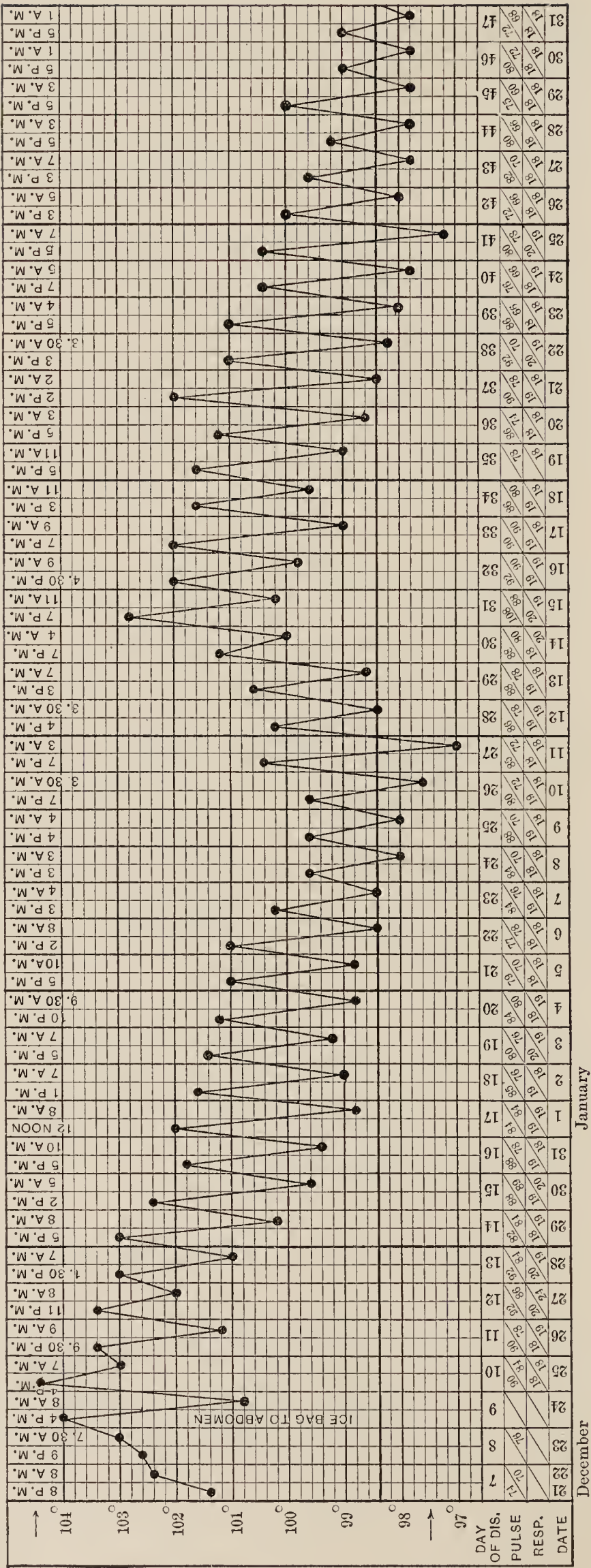
FIG. 299.



Mild typhoid fever. Gradual ascent. (Original.)

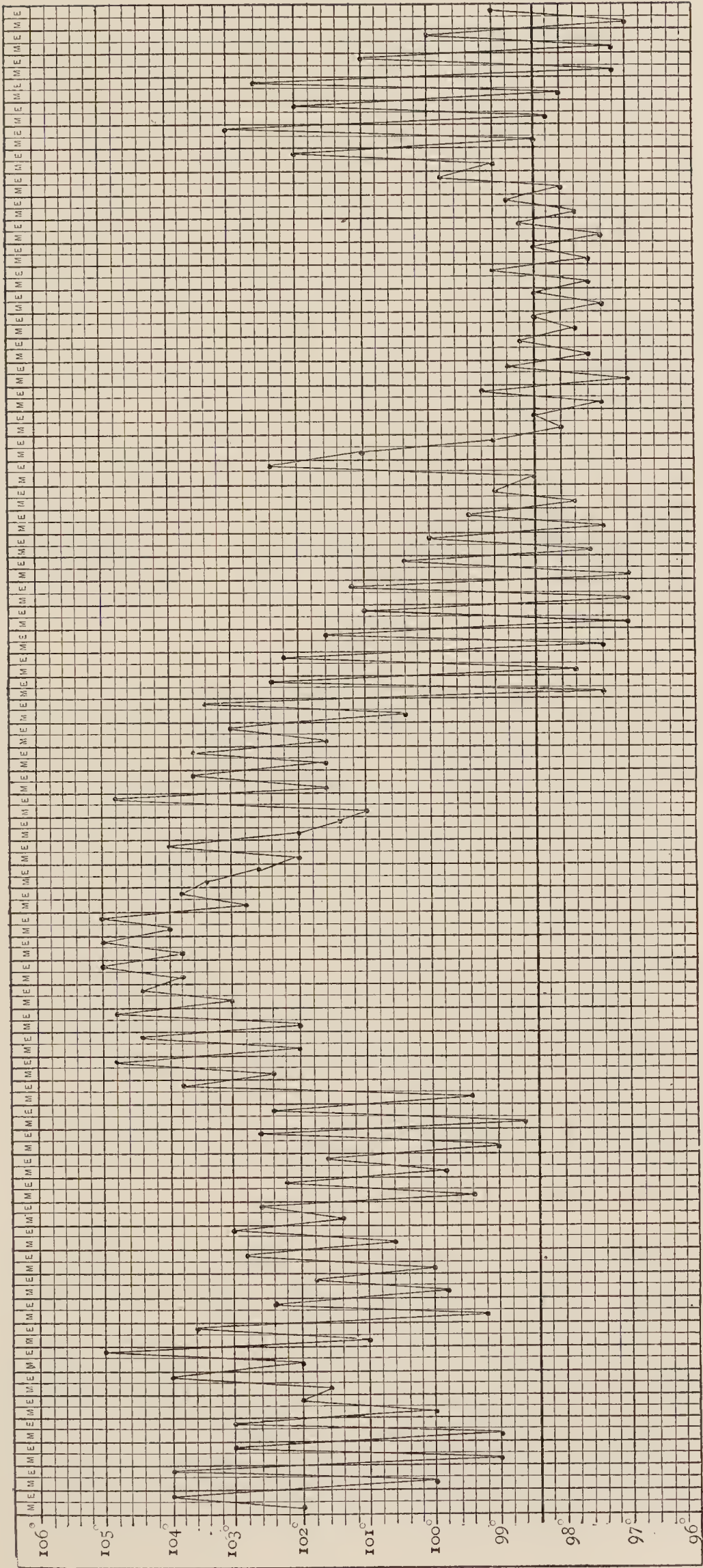
While the disease may begin abruptly, a gradual onset is so much the rule that it becomes an important factor in the diagnosis from other disease-conditions.

FIG. 301.



Typical chart, mild typhoid fever with an intercurrent relapse. (Original.)

FIG. 302.



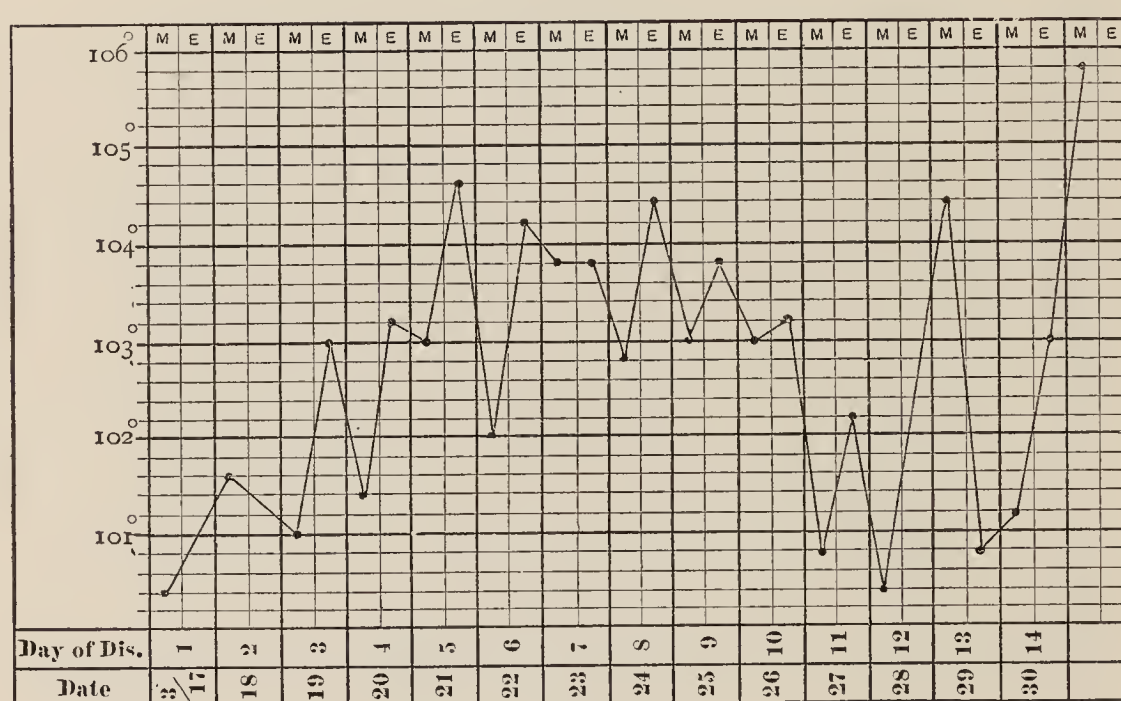
Protracted typhoid fever, not modified by antipyretics. Aberrant temperature during the first twenty days. Duration, sixty-three days.
Recrudescence on the fifty-fifth day. (Original.)

Enlargement of the spleen is a very constant symptom. It may be detected at the onset, increases up to the height of the fever, subsides during convalescence, but recurs during a relapse. It covers a percussion-area in the left hypochondrium of four to eight finger-breadths.

The *temperature-curve*, when not modified by treatment, shows a gradual ascent during the first four or five days of the disease, with morning remissions. The temperature rises a degree or two in the evening and falls half a degree or a degree in the morning. This "step-ladder" ascent is characteristic. By the end of a week a temperature of 103° , 104° , or 105° F. is reached, and it remains continuously high, with slight morning remissions, during the second and less frequently during the third week. In the third or fourth week the morning fall of temperature gradually becomes greater, and by the end of the week the temperature sinks below the normal in the morning.

The temperature in mild cases may never rise above 103° F. at any time, and most of the time varies between 100° and 102° ; or it may be 104° from the start; more frequently during the second and third weeks there are marked oscillations of the temperature—a sudden fall from 104° to 101° , or a rise from 103° to 105° or 106° . Hyperpyrexia is a temperature above 105° .

FIG. 303.



Grave typhoid fever. Death. M., aged twenty-two years. Ataxic symptoms. (Original.)

The *pulse* is full, and in favorable cases slower than the pyrexia would lead one to expect. It is more frequently under 110 than over 120. In the second week it is markedly dicrotic.

The *heart-sounds* are unchanged apart from complications, but in the second and third weeks the first sounds often are feeble, indicating heart weakness. A pulse of 120 or more is a graver sign in typhoid fever than in other diseases. Therefore, when it becomes very frequent and feeble, the extremities cool, and the lips bluish, the outlook is gloomy.

The *urine* is at first scanty and high-colored. A slight degree of febrile albuminuria is not uncommon, and in rare cases the whole force of the poison seems to be spent upon the kidneys, the urine containing,

besides the usual blood and casts, biliary coloring-matter. In conditions bordering on coma the patient may have retention of urine, or, on the other hand, he may pass it involuntarily. To obtain the *diazo reaction* of Ehrlich two solutions are necessary. The first (*a*) consists of 2 grammes of sulphanilic acid, 50 c.c. of hydrochloric acid, and distilled water 1000 c.c. The second (*b*) consists of a 0.5 per cent. solution of sodium nitrite. These solutions are kept in separate bottles. 50 parts of solution *a* and 1 part of solution *b* are poured into a test-tube and an equal volume of urine added. The test-solutions and urine are now thoroughly shaken and then carefully overlaid with 1 c.c. of ammonia. At the junction of the two a pink or ruby ring develops. Upon agitation the foam on the top of the mixture is also colored red. Normal urine gives a light-brown ring. This reaction is helpful in diagnosis, but may occur in acute phthisis, tuberculous meningitis, and other diseases. According to Pepper, it is rarely absent in measles. The reaction is fairly constant in typhoid fever after the first week.

The *respiration* in uncomplicated cases increases in frequency with the rise of temperature. It usually ranges between 24 and 36. The slight bronchitis present in the beginning in most cases causes no trouble; sometimes it lasts throughout and contributes to the tendency to hypostatic congestion, which is always present. The physical signs are those described elsewhere in these conditions.

The *nervous symptoms* are often very prominent. In mild cases they consist of hebetude and nocturnal delirium, or they may be absent altogether. Usually, however, by the beginning of the second week, there is some mental confusion, with nocturnal delirium. In more severe cases, and later in the disease, the delirium is of a low muttering character, with more or less continuous hallucinations of sight and sound. The patient can be roused by a question and makes an intelligent answer, but speedily lapses into semi-consciousness. Picking at the bed-clothes or efforts to catch imaginary objects are very common. Sometimes the delirium is wild and noisy, and the constant presence of someone is needed to keep the patient from getting out of bed. Patients have jumped out of windows, or run long distances before being captured. Rarely the delirium has been so active as to simulate acute mania. Stupor may alternate with delirium. Rarely the patient lies with wide-open eyes, apparently staring fixedly at some object, but really unconscious (coma-vigil).

In ataxic cases the patient has marked twitching of the tendons, and jactitation. He is wakeful and restless, wearing himself out. The hands and lips tremble, and he keeps muttering to himself all the time.

Convulsions are rare, but may occur in children. Sometimes there are considerable hyperæsthesia and tenderness along the spine.

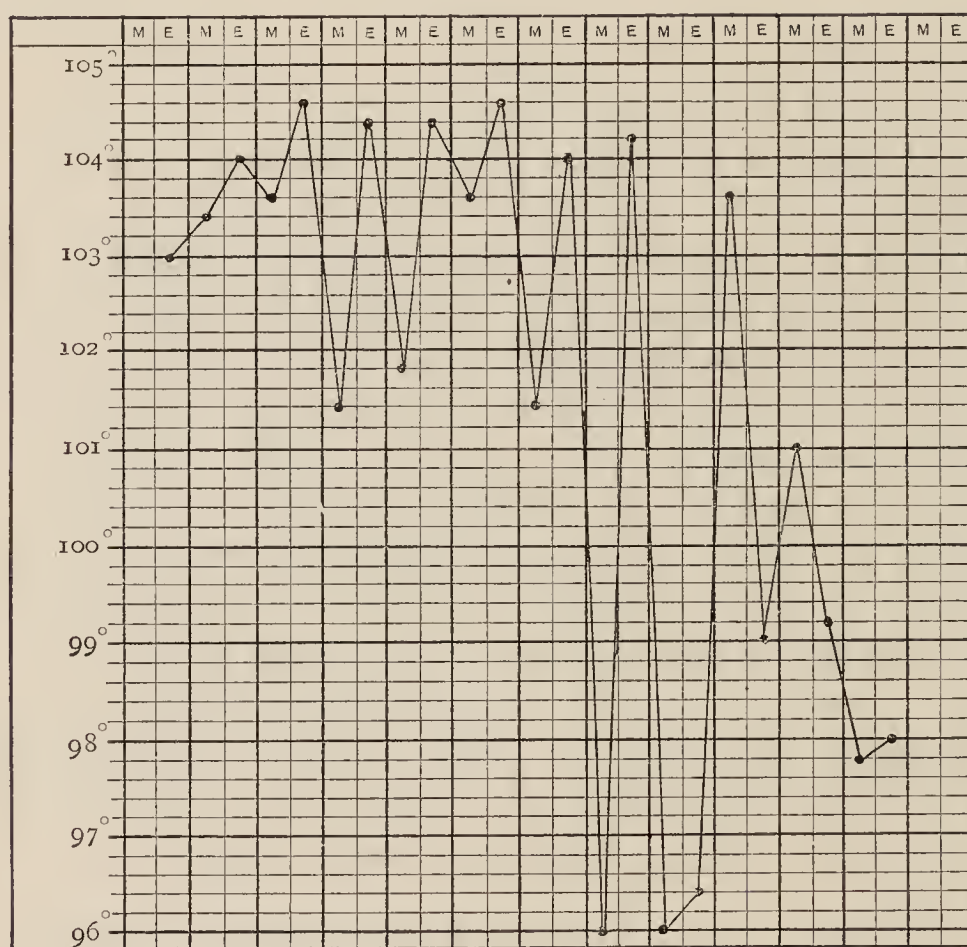
The severity of the nervous symptoms depends upon the habit of the patient as well as upon the height of the temperature and gravity of the disease. In children and neurotic individuals they may be pronounced with only moderate fever.

On the seventh or eighth day the *eruption* appears. It consists of small, very slightly elevated, rose-colored papules, which disappear upon

pressure and come out in successive crops, each papule lasting three or four days. The spots are most common over the abdomen and back, but are occasionally found elsewhere. They are usually few in number, a half-dozen or dozen, but sometimes the eruption is very copious, especially in severe cases. Sometimes it is wholly absent.

During the latter part of the second week, and throughout the third week, the symptoms are apt to be intensified. The temperature remains high or even reaches a higher point. Delirium is more decided and constant. The heart grows weak and the pulse increases in frequency. Some degree of hypostatic congestion of the lungs is usual. Diarrhœa may be troublesome; intestinal hemorrhages, announced by sudden fall of temperature and symptoms of collapse, may occur. Tympanites may become so great as to interfere with respiration and circulation. This is the period when ulceration of Peyer's patches in the intestine is deepest, and when perforation is imminent. There is rarely any desire for food, though it is taken and assimilated. Nausea and vomiting are rare. The tongue is dry, brown, sometimes glazed and fissured, and sordes often collect on the teeth.

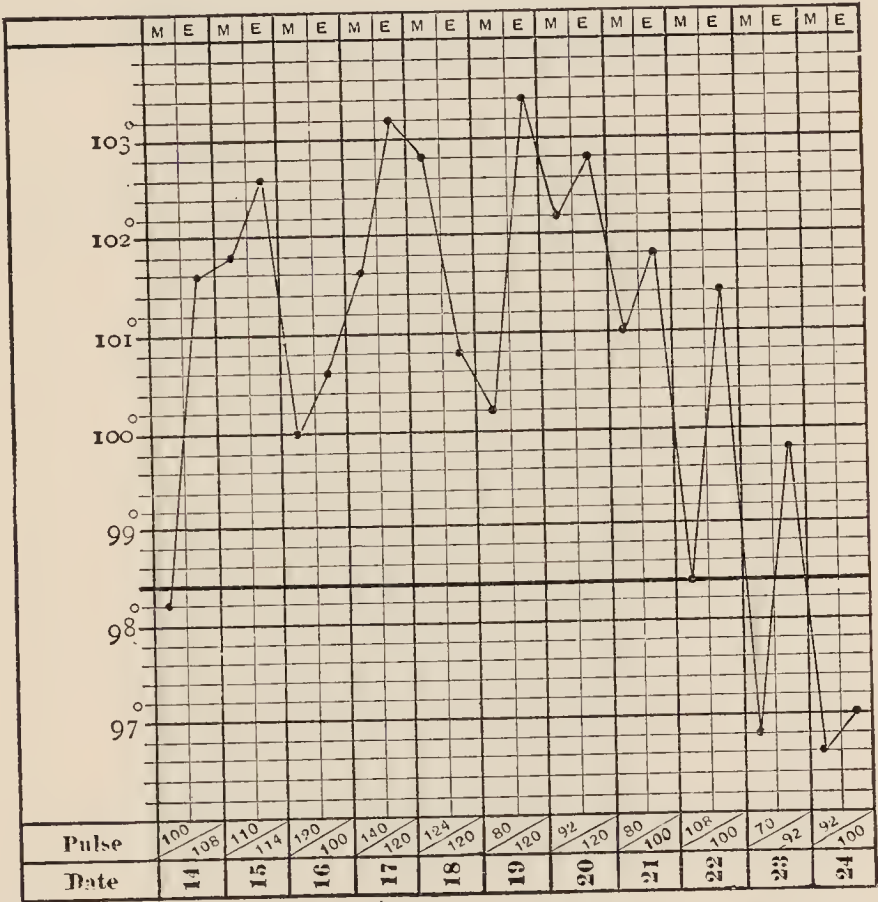
FIG. 304.



It may be interrupted by a relapse, in which the original symptoms are reproduced, with high temperature, but the duration is shorter.

Varieties. It is now well known, as Osler forcibly states, “that typhoid fever is no more primarily intestinal than is smallpox primarily a cutaneous disease.” Studies in bacteriology, promoted especially by Chiari, Flexner, Kraus, Nicholls, and others, enable us to divide the infection into three varieties: 1. Typhoid fever with intestinal lesions, as described above. 2. Typhoid fever with general infection or typhoid septicæmia, in which the symptoms are entirely those of an infection, and

FIG. 305.



Course of temperature in a relapse beginning on the twenty-sixth day.
First attack mild. (Original.)

the diagnosis must rest upon the serum reaction and culture-methods. 3. Typhoid fever with more intense infection of other organs than the intestines. The lungs, the spleen, the kidneys, and the cerebrospinal meninges are the structures invaded, so that we may have a pneumo-, nephro-, spleno-, or cerebrospinal typhoid.

Varieties are also based upon the severity of the disease; hence we have the abortive, ambulatory, and grave forms.

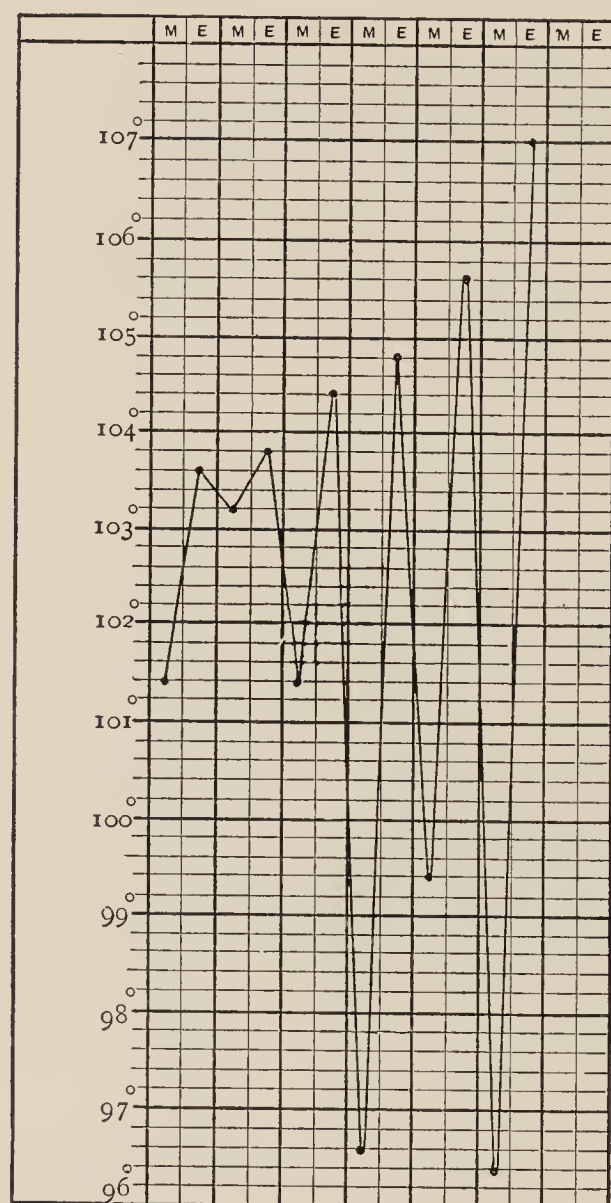
The *abortive* form is so named because of the abbreviated course of the disease. The symptoms are sufficiently well marked to make the diagnosis clear, but the type is mild, and in a week or two convalescence is established. In rare instances an afebrile form with intestinal symptoms and eruption is seen.

In the *ambulatory* form, commonly called “walking typhoid,” the patient, from ignorance of the gravity of his ailment or from apparent necessity, keeps at his work until weakness and incessant headache lead him to consult a physician in his office or at a dispensary. He may then

be well into the second week of the disease. The majority of such cases prove fatal.

Grave forms are due to especial severity of some symptoms or group of symptoms, such as hyperpyrexia; profound stupor, coma, or intense ataxia; inability to take or retain sufficient nourishment; profuse diarrhoea and intestinal hemorrhage; great adynamia with weak heart and a tendency to cyanosis. In other cases the gravity of the disease results from the existence of complications.

FIG. 306.



Grave typhoid fever. Daily rigors. Died on nineteenth day. No complications. (Original.)

In the *malignant* form either the dose of the poison has been large or the organism is very weak, or both, the result being an acute toxæmia; this is not so common as in scarlatina and typhus fever.

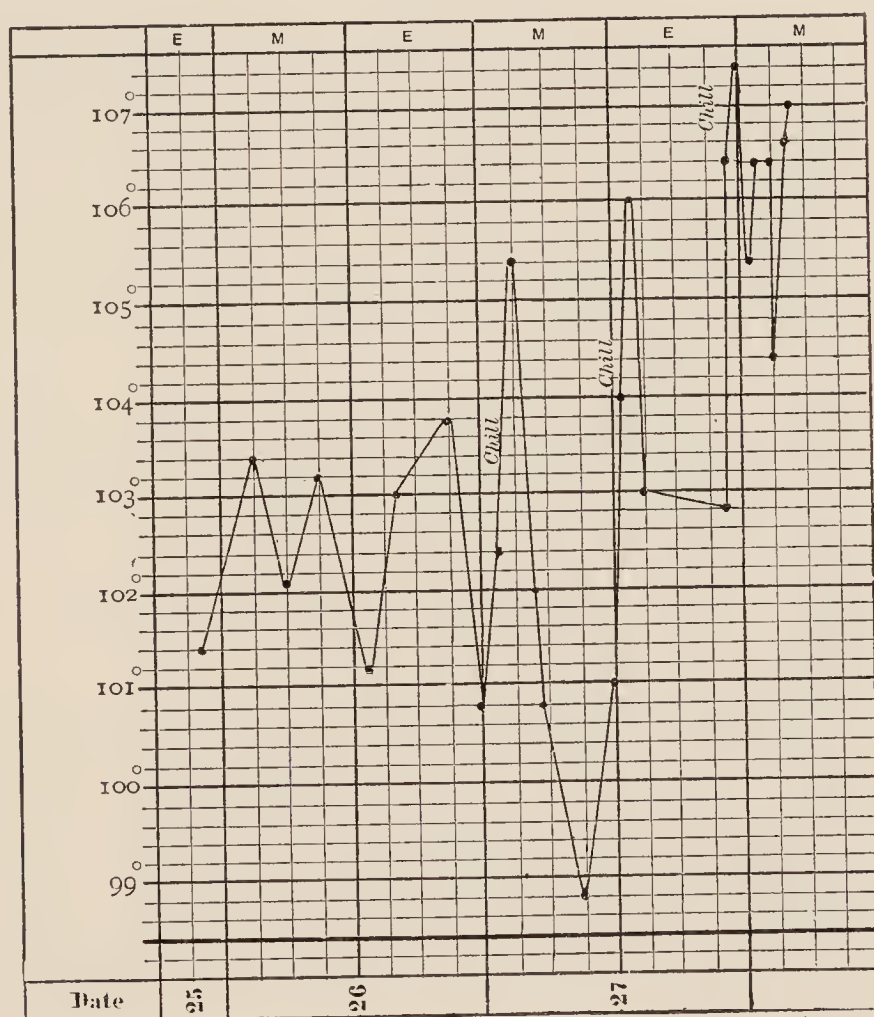
In the *pulmonary* form the onset may be so obscured by severe bronchitis or lobar pneumonia that the primary disease is not suspected at first. Severe bronchitis seems to be more common in children. Chill and initial high temperature are common in these cases.

Typhoid Fever without Intestinal Lesions. This rare form may present the typical clinical symptoms of typhoid, or may be of spleno-typhoid type, or of nervous type with extreme intoxication. The first type is rare. The second type, described by Eiselt, is characterized by an excessive enlargement of the spleen, with local inflammation and remit-

ting fever. In the third the symptoms of the typhoid state with subcutaneous and visceral hemorrhage occur. Jaundice is more or less common.

Complications and Sequelæ. Typhoid fever may be accompanied by a number of complications, the most frequent and important being severe laryngitis, bronchitis, hypostatic congestion of the lungs with œdema, and true lobar pneumonia; bed-sores; parotitis; phlebitis, especially of the femoral vein; peritonitis from perforation of the bowel; jaundice; cholangitis; meningitis, acute mania; myocarditis; periostitis and osteitis. Sequelæ are not frequent. Sometimes, however, the foundation is laid for permanent ill health. There may be impairment of the senses, mental weakness, and even insanity. Paralyses, neuritis, hyperæsthesia, chorea, and epilepsy are occasional sequels.

FIG. 307.



Renal typhoid. Nephritis on the twenty-fifth day. Course of temperature during three days preceding death. (Original.)

Examination of the Blood. The infection is due to Eberth's bacillus, *Bacillus typhosus*. The bacillus is found in colonies in the spleen, liver, mesenteric glands, kidneys, and intestines. It is also found in the feces and rarely in the urine. It may be seen in the blood. It may be recognized by staining methods, although rarely. It has been isolated from the blood successfully by culture-methods. The results of Kühnau and Gwyn show that in 20 to 25 per cent. of the cases, whether severe or mild, bacilli may be obtained. Gwyn had 8 positive cases, some of which were very mild infections. Richardson found bacilli very constantly in the spots (5 out of 6 cases).

Morphology. It has flagella 3 to 5 times as long as the bacilli. It stains with the anilines, best with Löffler's method.

Serum Diagnosis. This method of diagnosis has been more successfully employed in typhoid fever than in any other infection. The methods have been previously described. The agglutinative reaction takes place as early as the eighth day, rarely as early as the third day, but sometimes not until the fifteenth or twentieth day, and may even be delayed until convalescence is established. By this means typhoid fever can be distinguished from the infection due to the bacillus of Gärtner (*Bacillus enteritidis*).¹

The paracolon bacillus infection, as shown by Gwyn,² who studied a case which resembled typhoid clinically, does not give this reaction. Influenza and Malta fever, and forms of tuberculosis can also be distinguished from typhoid fever by this method.

Leucocytosis. Typhoid fever is one of the infections in which leucocytosis does not occur. In a differential count some variation from the normal is seen. The large mononuclear and transitional forms are relatively increased; the polynuclear neutrophils are decreased. The absence of leucocytosis aids in distinguishing typhoid fever from various septic fevers and acute inflammations. On the other hand, if leucocytosis occurs in a case of typhoid fever, an inflammatory complication or mixed infection should be suspected. Perforation and peritonitis are attended by leucocytosis.

In addition to the absence of leucocytosis we find, after the second or third week, gradual reduction of the red cells, and by the time convalescence is established, a marked anæmia develops. Both the red cells and the hæmoglobin are reduced.

Culture-methods. The bacillus can be isolated from the blood, the stools, and the urine.

Bacteriological Diagnosis. Recently bacteriologists have been successful in isolating the typhoid bacillus from the blood, the stools, and the urine. Unfortunately, the methods are too complicated for clinical work. P. H. His, Jr., recovered *Bacillus typhosus* and distinguished it from members of the colon group by a combined plate and tube method.³ For differentiation of the typhoid from the colon bacillus the method of Proskauer and Capaldi may be used.

Mark Richardson isolated bacilli in the *urine* of about 22 per cent. of the cases of typhoid examined. They were present in large numbers and in pure culture. They appeared late in the disease, and persisted into convalescence. The bacilli were always associated with albumin and casts. After disinfection of the meatus the urine is passed in two portions into sterilized test-tubes. The second portion is used. It is immediately plated upon plain agar. At the end of twenty-four hours the characteristic colonies appear. Richardson relies upon the active motility of the

¹ See Lancet, January 15, 1898.

² Bulletin of the Johns Hopkins Hospital, 1898, vol. ix., No. 84.

³ P. H. His, Jr., "On a Method of Isolating and Identifying *Bacillus Typhosus*," etc. Journal of Experimental Medicine, vol. ii., No. 6, p. 677.

bacilli, which are set free in a typhoid colony by scarring with a platinum needle to distinguish them from the colon bacilli. He also used the dry serum reaction test.¹

Gwyn had twelve cases in which the bacilli were present in enormous numbers, all in pure culture. In one the diagnosis was made from the urine, the Widal reaction being absent at the time. In two others the bacilli were recovered from the urine before the development of the Widal reaction. They were present in practically the same percentage as in Richardson's series. When one calculates, as in one case, 500,000,000 bacilli per cubic centimetre and sees the bacteriuria persist for weeks and years, the importance of the urine as an infective agent is apparent.²

Richardson and Steele in my wards at the Presbyterian Hospital have recovered the bacillus from the sputum of cases of typhoid fever with coincident bronchitis and pneumonia. The pneumococcus and the influenza bacillus are generally associated, and the sputum is hemorrhagic.

Diagnosis. A typical case of typhoid fever ought not to be mistaken for any other infection, but atypical cases are numerous.

New Diagnostic Sign of Typhoid Fever. Simon Baruch writes as follows: "As soon as the patient shows a rectal temperature above 102.5° in the morning and 103° in the evening for three successive days, especially if this be accompanied by headache, dulness, or apathy, he is placed in a full bath at 90° , which is reduced to 80° , with constant friction over the body. In three hours, the temperature still being above 102.5° , he receives another bath 5 degrees cooler. This is repeated until the temperature of the bath is 75° . If one or more of these baths fail to reduce the rectal temperature 2 degrees in half an hour, the diagnosis of typhoid fever is almost certain, and the bath-treatment is continued. The resistance of the rectal temperature to a bath of 75° for fifteen minutes with friction, is an almost certain sign of typhoid fever."³ Baruch considers that the diagnosis of this disease should no longer be obscure, even in the first days of its course.

Local Infections. The most common sources of error are a hurried diagnosis and a willingness to accept a demonstrable local affection as sufficient to account for the condition. In this way the significance of bronchitis, pneumonia, and diarrhoea is overlooked. In the symptomatic form there will almost always be found a history of gradual onset and a degree of fever and prostration greater than should attend the purely local affection. Moreover, in the bronchitis and pneumonia which are a part of typhoid fever, there may be found tenderness with gurgling in the right iliac region, enlargement of the spleen, and epistaxis, to aid in the diagnosis; while in cases in which the diarrhoea is misleading, bronchitis, enlargement of the spleen, and epistaxis may coexist.

Examination of the blood, extended over a period of several days, is necessary to exclude the *æstivo-autumnal* type of malaria, which often resembles typhoid fever. The Widal test, the absence of leucocytosis,

¹ M. W. Richardson, "On the Presence of the Typhoid Bacillus in the Urine." *Journal of Experimental Medicine*, vol. iii., No. 3, p. 349.

² Johns Hopkins Hospital Bulletin, April, 1899.

³ New York Medical Journal, September 2, 1893.

and the bacteriological examinations are essential methods for positively excluding the simulative infections, the consideration of which follows.

Appendicitis is more likely to be mistaken for typhoid fever than the converse. There is usually a history of constipation, though the occurrence of several inadequate movements a day may conceal the fact that there is a fecal accumulation. In appendicitis the onset is more abrupt and the local symptoms are more pronounced than in typhoid. Pain and tenderness are prominent in appendicitis, and while they may be general over the abdomen at first, they are found to be more acute in the iliac region and in the loin. Here, in place of gurgling, we find some increase of resistance on palpation, and a relatively dull note—a wooden sort of tympany—or there may be a demonstrable tumor. The patient lies with the right leg drawn up, has moderate fever, and vomiting. In fact, the attack is often introduced by chilliness and vomiting. Headache is not a prominent symptom, while bronchitis and enlargement of the spleen are absent. Leucocytosis is a valuable sign much insisted upon by the surgeons, while, of course, the presence of the Widal reaction favors typhoid fever.

Acute right-sided *salpingitis* simulates typhoid fever. It is distinguished by the history of a preceding vaginitis, endometritis, or abortion, and by the absence of diarrhoea, of enlargement of the spleen, and of the characteristic eruption. A digital examination through the vagina discovers the womb pressed to one side and fixed, and a tender mass blocking up the pelvis.

Simple continued fever is distinguished from typhoid fever of a mild type principally by the absence of bronchitis, of enlargement of the spleen, of epistaxis, and of the characteristic eruption of typhoid fever. In simple continued fever constipation is more common than looseness of the bowels, and gurgling is absent.

Typhus fever is distinguished by its sudden onset, the besotted expression of the face, with reddened eyelids and small pupils, the absence of abdominal symptoms, and the appearance on the fourth day of macules, which are subsequently converted into petechiæ. It is of shorter duration, and terminates very abruptly by crisis.

Relapsing fever differs from typhoid fever in its sudden onset with chill, pain in the epigastrium, but absence of abdominal symptoms and eruption; in the absence of marked nervous symptoms, in spite of the high fever; the short duration and termination by crisis, and the characteristic relapse at the end of a week. The conclusive test is the finding of spirilla in the blood.

Acute tuberculosis of the lung at times closely resembles typhoid fever. In both the onset is gradual, with cough and fever. In the former, however, the bronchial symptoms are more prominent, there are apt to be recurring chills and sweats, the temperature is remittent and irregular, emaciation is rapid, and constipation instead of diarrhoea is the rule.

In *peritoneal tuberculosis* there is persistent, diffuse pain in the abdomen, and the belly is swollen. If effusion occurs, the physical signs disclose its presence. The temperature is irregular and may be below normal; nervous symptoms comparable to those of typhoid are wanting.

Meningitis before the stage of effusion exhibits exaggeration of the reflexes and marked hyperæsthesia. There may also be muscular rigidity. The patient is restless, easily annoyed, and "fussy" about things that would be unnoticed by a typhoid patient. Vomiting is often present, whereas it is rare in typhoid fever. The temperature does not maintain so high an average range as in typhoid fever, and is subject to oscillations. The pulse varies greatly, and may be irregular. Leucocytosis is present, and cultures from "swabs" of the nose and nasopharynx or the cerebrospinal fluid may establish the diagnosis.

In *septic meningitis* the headache and vomiting are more persistent, the bowels are confined, and the abdominal walls are retracted. There may be double optic neuritis. In *tuberculous meningitis* the knee-jerk and other reflexes are variable, irregularly absent or present. In typhoid fever they are always present. In the former, choroidal tubercles may be seen with the ophthalmoscope. (See Eosinophile, Chapter XXXIX.) Leucocytosis is present in all forms of *tuberculosis*; in typhoid it is absent.

Typhoid fever must not be confounded with *trichinosis*; the peculiar muscular pain and œdema do not occur in the former. *Uræmia* may simulate typhoid fever when it becomes chronic; but the age, the character of the urine, and the cardiovascular symptoms are diagnostic, and, in the absence of the specific typhoid symptoms, render the diagnosis easy.

Mountain fever is an infection which has been described as peculiar to the mountains of our Western States, characterized by a continued fever with intestinal symptoms not unlike those of typhoid fever. Irregularity of the temperature range and the occurrence usually of constipation rather than diarrhœa make it difficult to separate the infection from typhoid fever on the one hand and from forms of malaria on the other. Recent observations of Woodruff, who studied the serum reaction in a large series of cases, show conclusively that the infection is typhoid fever, confirming the prior observations of Hoff, Smart, and Raymond.

Paratyphoid Fever. This infection is due to a bacillus which is intermediate between the typhoid and the colon bacillus.

The symptoms are like those of typhoid fever in the larger number of cases. In another, but smaller group of cases, the symptoms are those of gastro-enteritis with fever. The first form corresponds closely to typhoid fever in all its incidents, including the etiology and the occurrence of relapses. It is usually of shorter duration, premonitory symptoms are absent, prostration is early, myalgia is more marked, and the temperature rises more rapidly. In the gastro-intestinal form the temperature rises rapidly, after a chill, diarrhœa supervenes at once, vomiting and epigastric pain are marked, while prostration is the dominant feature. The diagnosis can only be established by the serum test. Cultures from the blood, urine, or stools would prove positively the presence of this infection.

In using the serum test, both species of the paratyphoid bacilli must be employed, and an agglutination should not be expected except in high dilution. If the blood fails to agglutinate the typhoid bacillus or agglutinates it only in very low dilutions, typhoid fever is absent.

Yellow Fever.

The infection which we are about to consider is the latest of the epidemic and contagious disorders for which a definite causal micro-organism has been discovered. It is an acute, specific, contagious, miasmatic disease, endemic and epidemic on the tropical and subtropical shores of the Atlantic Ocean, characterized by a sudden onset, a duration of a week or less, a characteristic facies, a fall in the pulse-rate preceding a fall in the temperature, and by albuminuria, jaundice, and vomiting, with a tendency to hemorrhages. The specific micro-organism is *Bacillus icteroides* described by Sanarelli. Sternberg asserts that his bacillus X is identical, and hence claims priority.

Yellow fever is endemic in Havana and other seaport cities of Cuba, and in Rio Janeiro, Brazil. From these centres it is liable to become epidemic and to be carried in ships and by persons and clothing to other places. In this way epidemics have developed in the seaports of the United States, especially on the shores of the Gulf of Mexico, but sometimes as far north as Philadelphia and New York. The disease becomes epidemic in the hot season and disappears upon the appearance of frost.

In countries in which the disease is endemic it is the custom to regard the native children as immune. John Guitéras, however, is strongly of the opinion that the disease is kept alive between epidemics by mild cases among these children. He has also shown that it prevails among white children before it becomes epidemic among adults.

The period of *incubation* varies from a few hours to two weeks. Guitéras states that the cases in which it extends beyond the seventh day are exceptional.

The *invasion* is abrupt, and occurs usually in the night. It is marked by chilliness oftener than by a decided chill. The temperature rises rapidly to 102° to 103° or 104° F., not often higher in favorable cases. The *pulse* is correspondingly increased in frequency at first, but very commonly begins to fall before the temperature, so that later the pulse is relatively slow. The *face* is peculiar and characteristic—it is flushed and somewhat swollen; the edges of the eyelids are reddened; the eyes are watery, glistening, and slightly but distinctly tinged with *yellow*; the pupil is small and brilliant. Guitéras says:¹ “The appearance of the face is often sufficiently characteristic on the first day of the disease to warrant a positive diagnosis.” “The early manifestation of jaundice is undoubtedly the most characteristic feature of the facies of yellow fever.” He also says that these phenomena are often better observed at a slight distance than on close inspection. The *tongue* is large, moist, and coated with white fur. The stomach is irritable and the epigastrium tender. Nausea with repeated vomiting occurs. The fluid is at first of a light greenish-yellow, subsequently becoming decidedly bilious. The bowels are constipated.

The *urine* almost invariably contains albumin at some time during the first three days. The albuminuria may last only a short time and may

¹ “Report of the Surgeon-General of the Marine-Hospital Service, 1888”; Keating’s Cyclopædia of Diseases of Children, 1889, vol. i.

be found only in the evening; the amount of albumin is sometimes very large, and abundant blood- and tube-casts are found. The nephritis subsides rapidly, without leaving traces. The urine is acid in reaction and scanty in amount. It is sometimes suppressed.

During this febrile period the patient complains of headache, pains in the back and limbs, and intense thirst. The mind, however, is usually perfectly clear. Contrary to expectation, Guitéras asserts that the nervous symptoms are perhaps more prominent in the adult than in the child. "The loquacity, the short-cut phrases and precipitate speech, the excitement, the show of indifference with unmistakable evidences of fear—all these, that are such prominent features of the disease in the adult, are absent in the young."¹

In from two to five days the temperature falls to or below normal, headache and pain in the limbs disappear, and the patient is cheerful and believes himself convalescent. In mild cases this is the fact, but in more severe cases the period of remission or stage of *calm* is followed in a few hours or at most a day or two by a return of symptoms. The jaundice deepens, vomiting becomes more urgent, and in adults is accompanied by much retching. The vomitus is bilious, streaked with blood, or thick and wholly black ("black vomit"); the temperature may equal or surpass that of the original paroxysm, or it may remain depressed. In any event the pulse is apt to be slow, often from 40 to 60. The urine contains albumin, blood, and casts, and there may be suppression, adding uræmia to the other toxæmia. Convulsions at this stage are usually uræmic. Hemorrhages may occur from any mucous surface. The gums are tender, swollen, and bleed easily. There may be epistaxis, hemorrhage from the ear, bowels, uterus, or vagina. Pregnant women miscarry. Ecchymoses also may form. Death may take place in coma or convulsions. If the patient lingers beyond the fifth or sixth day, he sinks into a typical typhoid state, with diarrhœa and marked adynamia, from which he may or may not emerge.

As in scarlet fever, the patient may be smitten down and die in a few hours from the time he was in apparent health. In other grave cases the temperature remains high, and rises instead of falls on the third or fourth day. The *duration* of the disease is from two to five or six days; if a typhoid state develops, it may last ten days or two weeks.

Complications are not common. Phlebitis and lymphangitis occur, and Guitéras says he has noticed hepatitis, insanity, and paralysis (probably from neuritis). Second attacks are extremely uncommon.

Examination of the Blood. *Bacillus icteroides* is a slender rod from $2\ \mu$ to $4\ \mu$ in length. It is ciliated and motile. It is found in more than half the cases, if Gram's method is employed.

Serum Diagnosis. Woodson and the Archinards have found agglutination to take place in a large proportion of cases of yellow fever. The blood, taken as early as the second day, gave a prompt reaction in from 75 to 80 per cent. of all cases. Dilutions of 1 to 40 were used, but reaction took place in dilutions as low as 1 to 50. Pothier and Lerch report successfully upon this reaction. Cultures from the blood produce

¹ Keating's Cyclopædia, loc. cit.

an organism which grows on ordinary media, does not coagulate milk, but ferments saccharine fluids.

Inoculation. Inoculation of dogs and monkeys produces a clinical picture similar to the original infection.

Diagnosis. Yellow fever is distinguished from *pernicious malarial fever* by the slow pulse, the characteristic facies, the early transient albuminuria, the early and deep jaundice, the absence of diarrhoea, the occurrence of black vomit, the tendency to hemorrhage, the clear mind, and the absence of enlarged spleen.

The diagnosis is based upon examination of the blood, the three important characteristics which Guitéras lays stress upon—the facies, the albuminuria, and the slowing of the pulse, with continuance or increase of the fever, and the usual diagnostic data of an epidemic and contagious disease. The affection must be distinguished from dengue and from various forms of malarial fever, especially the *æstivo-autumnal* infections. Jaundice rarely appears as early as the second or third day of the disease. Hemorrhages are rare in both dengue and malarial fever.

Malta Fever.

Malta fever is an infection that prevails in tropical countries. It is characterized by gradual onset and by repeated remissions of the *fever*. The alternating febrile and afebrile periods which characterize the disease continue from two months to two years. The most remarkable feature is the peculiar character of the temperature range, which consists of intermitting waves or undulations of fever of a distinctly remittent type. These periods of fever last from one to three weeks, followed by an apyretic period or a period of abatement lasting from two to ten days. The daily temperature range may be intermittent or remittent. The febrile course may continue six months or longer. During this time patients grow more and more prostrated, become anæmic, and usually suffer from constipation. Profuse sweats attend the decline of the daily range, and in many instances the spleen is enlarged. Neuralgias occur in various parts of the body; the joints become enlarged, and fibrous tissues may be the seat of inflammation. Hughes—who describes the disease most accurately—describes a malignant type lasting a week or ten days, and an undulatory type continuing for weeks or months. Indeed, the relapses are known to occur over a period of two years. The third is known as the intermittent type, in which there is a daily rise of temperature without other marked symptoms. The undulatory type is the most common. The infectious micro-organism is *Micrococcus melitensis*.

Diagnosis. A positive differential diagnosis is made from all forms of malaria by an examination of the blood, and from typhoid fever by the Widal test and the absence of *Bacillus typhosus* in the urine or stools of the suspected patient. The micro-organism has not been isolated from the blood, but the *serum reaction* is a valuable means of diagnosis. (See page 552.) This reaction is obtained as in cases of typhoid fever. The culture must be carefully selected. With a 1 to 10

or 1 to 50 dilution agglutination takes place when the serum of a patient with Malta fever is used. The serum of such a patient does not have

FIG. 308.

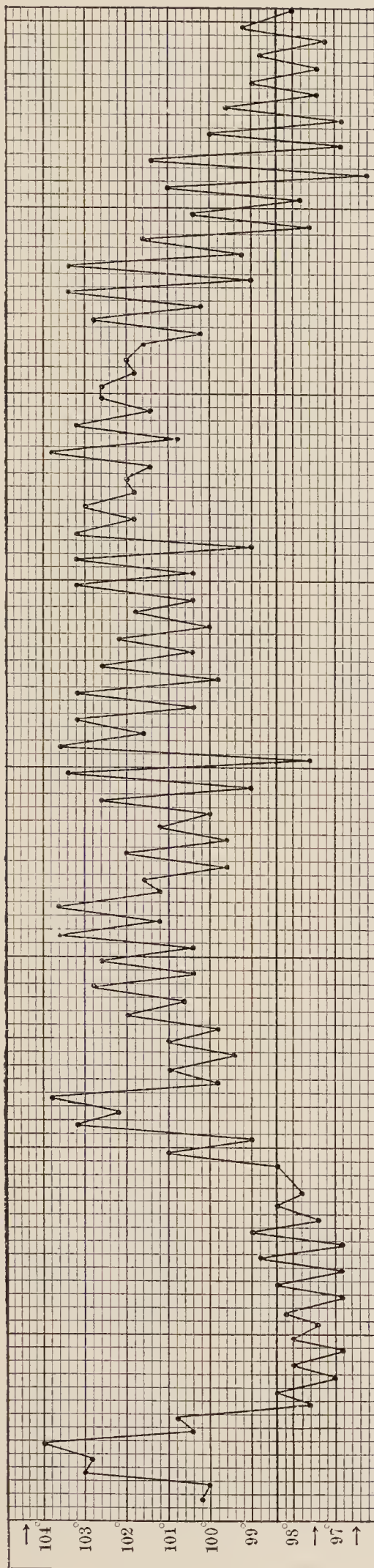
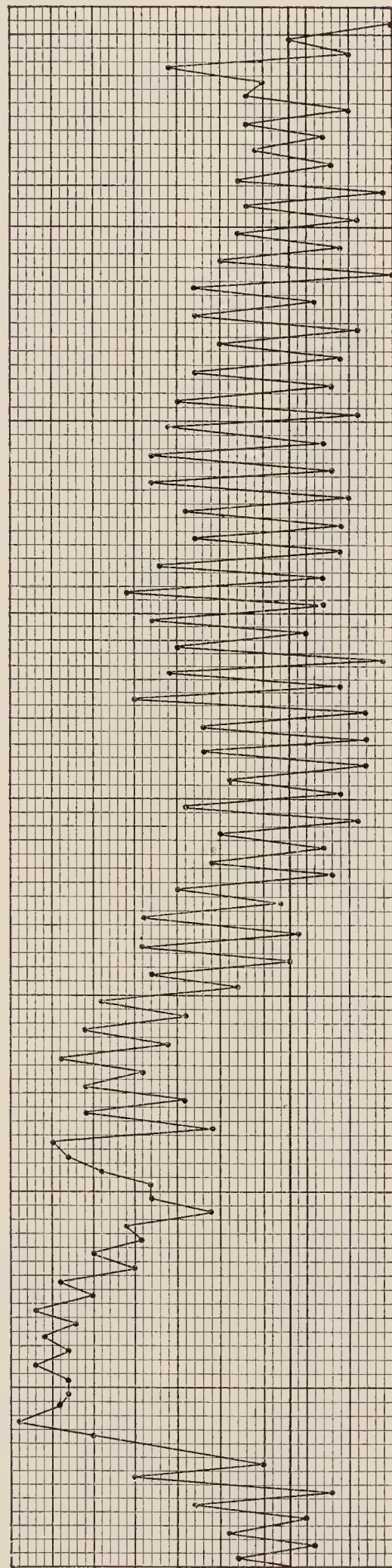


CHART SHOWING MORNING AND EVENING TEMPERATURES. TWO DISTINCT RELAPSES ARE SHOWN.



Malta fever. (MUSSEY and SAILER.)

any effect upon the typhoid bacillus nor upon other organisms. Aldrich states that the reaction first occurs about the fifth day.

BACTERIOLOGICAL EXAMINATION.

Gonorrhœal Infection.

Although the infection is usually limited to the genito-urinary tract, it is well known that the gonococcus may enter the blood and infect tissues elsewhere, causing a local inflammation. We therefore see symptoms due to the primary infection; symptoms due to infection of the genito-urinary organs by direct continuity and systemic infection. The primary infection involves the adnexæ of the genital organs in the male and the female. Salpingitis, metritis, and ovaritis in females, with the occurrence occasionally of peritonitis, arise from spreading by continuity. In both sexes, cystitis, ureteritis, and pyelitis occur. The infection is usually mixed. When the gonococcus invades the blood, symptoms of septicæmia or pyæmia arise. The infection may be rapid and fatal, and may terminate ten days after the primary lesion. The occurrence of such general infection is suspected when the history of the primary infection can be secured, and in addition the micro-organism can be recovered from the blood, as was successfully done by Thayer.

In other infections the joints become involved and we have the phenomena of gonorrhœal arthritis (see Joints), the course and symptoms of which are discussed elsewhere. Endocarditis may result from gonorrhœal infection, and can only be distinguished from other forms of endocarditis by the history and the finding of micro-organisms in the blood. Myocarditis (Councilman) and pericarditis may also occur.

Diagnosis. Thayer, Blumer, Thayer and Lazear have succeeded in recovering the gonococcus from the blood in this form of septicæmia. The blood is drawn from the median basilic vein with a sterilized syringe. It is mixed with melted agar and immediately plated. Cover-slip preparations of the colonies, if the case is gonorrhœal, will show the tinctorial and morphological characteristics of the gonococcus. (See Plate XIII.—C, Fig. 4.) The diagnosis is further established by finding the gonococcus in any purulent discharge, urethral or vaginal. (See Chapter XLV., Part I.—Exudates, etc.)

INFECTIONS RECOGNIZED BY THE EXAMINATIONS OF EXCRETIONS AND SECRETIONS OR BY THE PRODUCTS OF THE INFECTIOUS INFLAMMATION.

The following infections are disclosed by the examination of the products of the infection found in the inflammatory areas (pus); in the excretions and secretions of the body; in the sputa; in the vomitus; and in the feces, or in the urine. The reader should turn to the sections describing the method of bacteriological examination of pus, sputum, and secretions. The infections referred to are: *erysipelas*, *pneumonia*, *tuberculosis*, *influenza*, *cerebrospinal meningitis*, *diphtheria*, *septicopyæmia*, *gonorrhœa*, *glanders*, *cholera Asiatica*, *dysentery*, *bubonic plague*, *leprosy*, *actinomycosis*, *tetanus*, *trichinosis*.

Erysipelas.

The fever of this infection, particularly in a first attack, is very marked, and rises suddenly to a considerable height and may precede the eruption. It resembles the course of a pneumococcus infection.

Erysipelas is an acute, specific, contagious, and infectious disease, characterized by a sudden onset, with a bright-red eruption which usually begins on the face near the nose or mouth and spreads over the entire face and scalp. It is attended with burning heat of the skin and great disfigurement from swelling.

The specific cause of erysipelas is *Streptococcus erysipelatosus*. It is carried to a slight extent by the air, and still more in the discharges, especially those of the nose. Repeated attacks occur in persons with chronic nasopharyngeal catarrh, carious teeth, or a sinus. This infection is apt to attack persons with open wounds (surgical erysipelas), and puerperal women, producing in these cases sloughing and septicæmia. One attack does not protect against another; on the contrary, if there is any focus in which the streptococci linger, one attack actually predisposes to another.

The *period of incubation* is usually from three days to a week. On close inquiry a history of sore throat and some enlargement of the cervical lymphatics is usually found to have preceded an attack of facial erysipelas. The *invasion* is sudden and is marked by chill. The temperature rises to 104° or 105° F., and in the next two or three days may rise still higher. Coincidentally with the rise in temperature the portion of the *skin* to be affected burns, tingles, is tender to the touch, and may be reddened. The redness increases in intensity and extent, while the skin is swollen and slightly œdematous. The part of the face affected is usually the cheek in close proximity to the nose, less frequently near the mouth and ear. Vesicles and blebs often form when the inflammation is very intense. The redness disappears upon pressure, but quickly returns; sometimes it has a dusky, purplish hue.

A marked characteristic of the disease is its tendency to spread. In ordinary cases it involves one cheek, eyelid, and ear, and travels across the bridge of the nose to the other side. The inflammation is most intense while it is spreading; the advancing margin is raised, tense, and brawny; the line is thus sharply drawn between healthy and inflamed tissue. The loose tissue about the eyes swells enormously, both eyes are closed, the entire face swollen, red, and disfigured with vesicles and blebs here and there. Curiously the chin escapes. The redness and swelling begin to subside in the part first attacked, before the process has reached its height on the opposite side. As a rule, facial erysipelas does not extend beyond the face, the scalp and neck being spared. The scalp, however, is more frequently affected than the neck; occasionally erysipelas leads to extensive cellulitis of the scalp, with the production of a septic constitutional condition and much local sloughing. The submaxillary glands are more or less enlarged, sometimes so much so as to prevent the taking of solid food. On the body the eruption spreads over a greater extent than when primary on the face, hence its name, "the red runner." It may pass from

the heel to the thigh, and over the trunk, and may last for weeks. While the eruption is extending, the fever continues and is sometimes alarmingly high. The pulse is frequent and soft. Leucocytosis is present. Nocturnal delirium is not uncommon in severe cases, and nausea and vomiting are frequent. The bowels are usually constipated. The urine is high-colored, frequently contains a small amount of albumin, and actual nephritis sometimes occurs.

Prognosis. In favorable cases of facial erysipelas the process is at an end in a week or less ; it may be prolonged to two weeks, subsiding by crisis or lysis. Convalescence is usually rapid ; the vesicles or bullæ dry up into yellowish crusts, and the epiderm is shed in large or small pieces according to the intensity of the process.

Complications and Sequelæ. Pneumonia and nephritis are the most frequent complications. Meningitis, pericarditis, and endocarditis also occur. Erysipelas may extend inward and involve the fauces, pharynx, and larynx, producing œdema and death from suffocation. If the scalp has been involved, the hair falls out. The cervical adenitis may result in abscess ; chronic nephritis may occur. Otitis media occurs occasionally, and so do keratitis and abscess of the eyelids. On the other hand, erysipelas is credited with causing the disappearance of lupus, chronic eczema, and sarcomata.

Diagnosis. Bacteriological Diagnosis. Examination of pus, or of discharge from the nose or pharynx, will disclose the presence of the streptococcus. (See Plate XVII., Fig. 1 ; and Chapter XLV., Part I.)

Herpes zoster of the face and forehead is distinguished from erysipelas by the fact that vesicles appear first, followed by erythematous redness, and that they are limited by the median line, and are preceded and accompanied by sharp neuralgic pain, whereas erysipelas affects both sides of the face, and vesicles appear at the height of the disease ; the pain is much less in erysipelas.

Dermatitis. From dermatitis of various kinds it is distinguished mainly by the sharper febrile reaction, the raised border of the eruption, which begins on one side and spreads to the other. Erysipelas is rarely equally intense upon the two sides ; dermatitis frequently is. In the latter the skin often exhibits a rough surface, whereas in erysipelas the skin is smooth and shiny until vesicles appear.

Chronic *erythematous eczema* occurs in middle-aged and old persons, is afebrile, accompanied by little sweating but a great deal of itching, and runs a slow course.

Pneumonia. Pneumococcus Infection.

In typical cases of pneumococcus infection the course of the *fever* is of great diagnostic significance. Its sudden rise to a great height, preceded by a rigor, is of itself suggestive. During the succeeding days of the disease the morning and evening temperature vary but little. When associated with hurried respiration and the intoxication symptoms attending this infection, even though no physical signs are present in the lungs, pneumonia can reasonably be suspected. The termination of the febrile

PLATE XVII.

Fig. 1.



STREPTOCOCCUS—ERYSIPELAS.

Fig. 2.



STREPTOCOCCUS SEPTICUS.

Fig. 3.



STAPHYLOCOCCUS.

Fig. 4.



DIPHTHERIA-BACILLI.

Fig. 5.



TYPHOID-BACILLI.

Fig. 6.



TUBERCULOSIS-BACILLI.

course is characteristic of this disease. The sudden fall to normal or a subnormal temperature—known as the crisis—brings to an abrupt end the usually alarming symptoms.

Acute pneumonia, croupous or lobar pneumonia, is an infectious inflammatory disease excited by *Micrococcus lanceolatus* (*Diplococcus pneumoniae*, pneumococcus) involving the vesicular structure of the lungs, and followed by choking of the alveoli with the products of inflammation; it is attended by severe constitutional symptoms due to the toxins of the infecting organism.

Symptoms. Mode of Onset. The invasion of pneumonia is usually sudden, and is marked by a *chill*. The temperature rises rapidly, and may reach 104° or 105° F. in the first twelve hours after the chill. With the fever, the patient complains of severe headache and *pain* in the side, and has a short, quick cough and sometimes vomiting. The pulse is moderately accelerated, and the respiration either is or soon becomes very frequent. The face is apt to be flushed, and there may be a circumscribed red spot on the cheek. The skin is hot and dry. On physical examination, within the first twenty-four hours, a small patch of consolidation is detected, which may subsequently extend over a large area.

While this is the picture of an ordinary pneumonia in its early stage, all cases are by no means so clear. In some the course resembles that of a general fever in which the pulmonary disease is a local manifestation. In such cases there may be prodromata, consisting of headache, general malaise, a slight bronchitis, and digestive disturbance. Then follows the chill.

Central Pneumonia. The fever may be high for several days before there is discoverable consolidation of the lungs, and during this time cough may be wholly or almost wholly absent. The respirations increase gradually in frequency, and finally a well-marked pneumonia can be made out. It is customary to account for these cases by the supposition that pneumonia developed in the interior of the lung, and consolidation gradually extended to the surface. In some cases the patient presents no more definite symptoms for three or four days than high fever, intense headache, and moderately accelerated respiration.

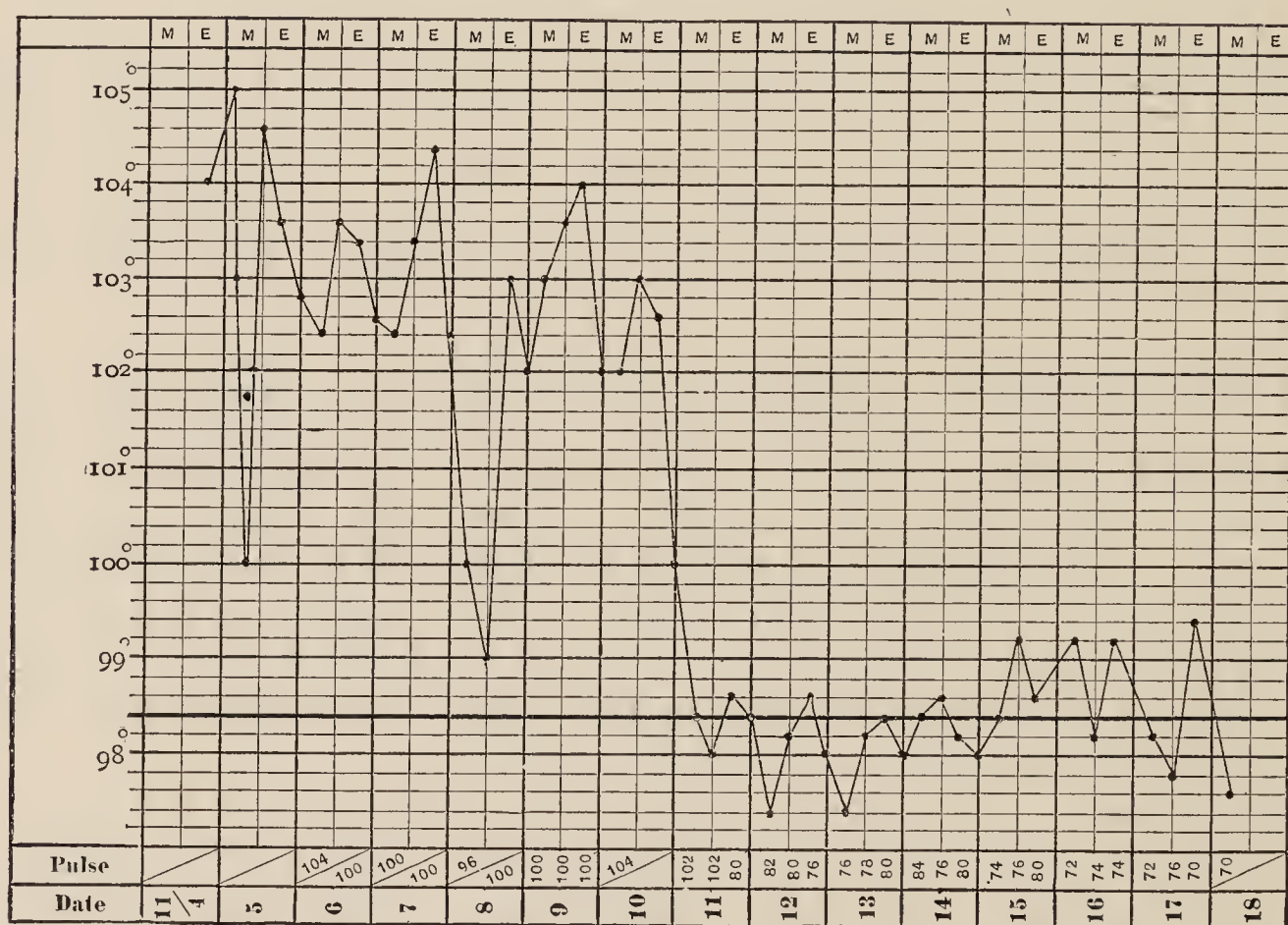
Later Stages. At the end of forty-eight hours, or, at the most, of four days, the patient is found lying in bed in the dorsal position, or on the affected side. The face is flushed, and countenance anxious, the respiration hurried, the *alæ nasi* play vigorously. The temperature varies little from the first day's rise; the chest pain has subsided, and the short dry cough is now attended by viscid expectoration. The respiration continues hurried, the pulse full and bounding. During this time the physical signs of consolidation continue and increase.

After a period of five to ten days the termination takes place by crisis, the pain in the chest abates, the cough becomes looser, and the expectoration more free, but the other symptoms persist. In addition, in some cases, delirium occurs, the pulse softens and becomes dicrotic, the urine becomes albuminous.

Respiratory Symptoms. Chest-pain, cough, hurried respiration of a peculiar type, and expectoration are characteristic. The *chest pain* is

sharp and stabbing or lancinating. It is increased by breathing. It is seated about the nipple or in the *axillary region*, at the angle of the scapula or below the diaphragm. Its seat always indicates the side affected. *Cough* is short and dry, smothered and painful; it soon becomes softer and painless as the expectoration becomes free. It may be absent in the feeble, in the aged, in alcoholic subjects, or in persons with brain disease, including insanity. Characteristic symptoms of pneumonia are the increased frequency and the type of the *respiration*. The rate in adults reaches 40, 50, or even 60 per minute, and in children 80 and 100 are not uncommon. The pulse, on the contrary, does not increase in frequency in the same proportion; hence the normal ratio of respiration to pulse of 1 to 4 becomes 1 to 3 or 1 to 2. Inspiration is short, expiration quick and often attended by an expiratory noise or grunt. The long pause may take place after inspiration instead of expiration. In children both are so short that unless the epigastrium is inspected, it may be difficult to distinguish the two.

FIG. 309.

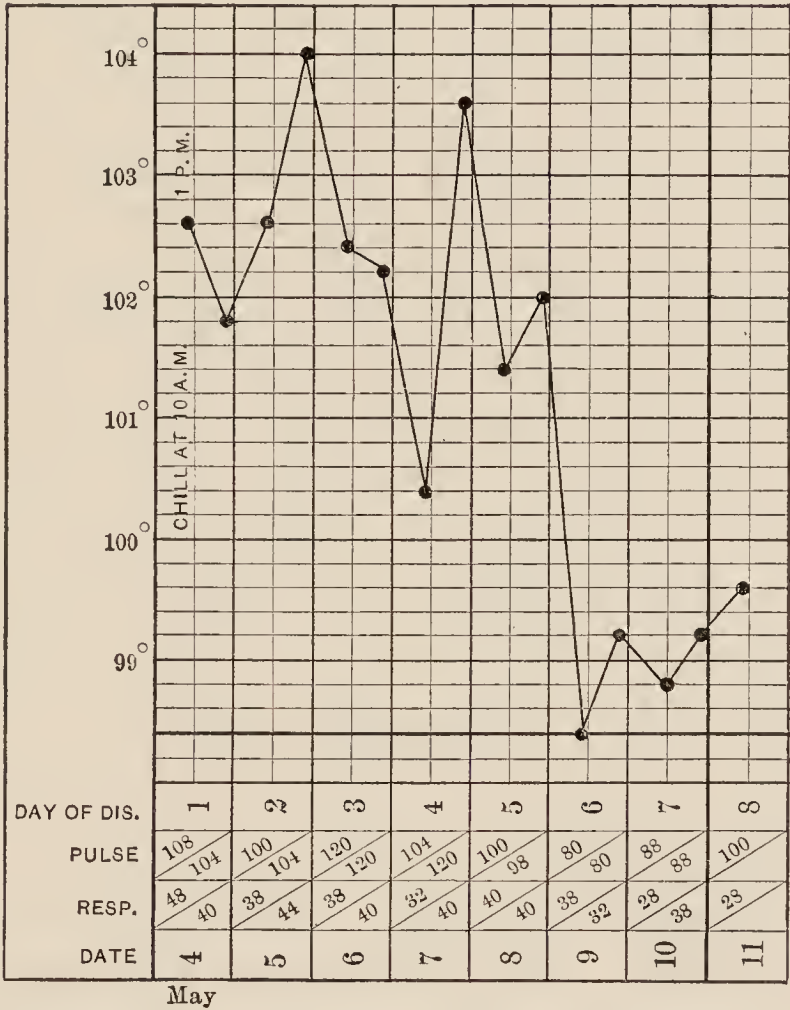


Pneumonia; sudden rise; termination by crisis; pseudocrisis on eighth day. (Original.)

In ordinary cases which run a normal course the cough is followed by *expectoration*, which is at first viscid mucus, but gradually becomes reddish brown from admixture of blood—the *rusty sputum* of pneumonia. This sputum is characteristic, almost pathognomonic. It is expelled with difficulty from the mouth, clinging to the lips or to the mustache. It can not be removed from the spit-cup by turning it upside down. It continues to be rusty until the crisis approaches, when it becomes purulent and is discharged with ease. In typhoid pneumonia it looks like prune-juice. (See Sputum.) It contains blood, alveolar epithelium, the specific micrococcus, and later, pus and small fibrinous casts.

Fever. The chill that precedes the fever is pronounced, and is always a warning to look for a pulmonic inflammation. In children a convulsion is rarely absent in frank pneumonias. During its occurrence the body temperature rises. In twelve hours it reaches 104° to 105° F. (See Figs. 309 and 310.) It remains at this point, obeying the laws of diurnal variation. The hot, dry skin, the parched lips, the dry tongue, the thirst, the anorexia, the hurried breathing, the occasional delirium, the loaded urine attest its presence. At the end of the third, or more frequently the

FIG. 310.



Pneumonia from first day ; pseudocrisis on fourth day ; crisis began on fifth. (Original.)

fifth, seventh, or ninth day, *crisis* takes place ; the fall is abrupt, and the normal or a subnormal temperature may be reached in from five to fifteen hours. *Pseudocrisis*, as the accompanying chart indicates, may precede the true crisis by twenty-four or forty-eight hours. The decline however, may take place by lysis. Protracted fever indicates delayed resolution or the occurrence of a complication.

Cerebral Symptoms. In some cases, especially in children, the onset of the disease may be marked by a *convulsion*. This is said to occur more frequently in apical pneumonias than in pneumonias of the base. Headache and delirium are so pronounced in some cases as to simulate meningitis. This is most likely to be the case in severe apical pneumonia in children, and in double pneumonia either in children or in adults. *Delirium* may occur during the height of the fever, and occasionally is maniacal. Nocturnal delirium may be a constant symptom in very grave cases. In drunkards it may simulate delirium tremens, and may be pronounced without much fever. In the later stages of grave or fatal cases a low form of delirium, with a tendency to coma, is common.

Heart and Pulse. The pulse is small at the time of the chill, but becomes full and bounding during the fever; later it may become dicrotic. Reference has been made to the pulse-respiration ratio. The pulse varies in frequency and in character with the type of the disease. In healthy adults it is rarely over 110. In the debilitated it may be very frequent, small, and feeble; in the aged, frequent and dicrotic. Extensive consolidations reduce the amount of blood in the general circulation, cause rapid action of the heart and a small pulse, and favor death with the heart in asystole.

The heart-sounds are clear. A murmur low in pitch is often heard in the mitral and pulmonary areas. The left ventricle acts forcibly. The pulmonary second sound is accentuated. If dilatation and failure of the right heart take place, the area of dulness may extend beyond the right edge of the sternum, an epigastric impulse be noted, turgescence of the veins in the neck become marked, but above all, the previously accentuated pulmonic second sound may become weak or disappear.

Gastro-intestinal Symptoms. Vomiting frequently occurs in children at the onset, and both in them and in adults may persist and mask pulmonary symptoms. The appetite is lost. The tongue is furred, and may become dry and brown. The bowels are constipated except when complications occur. The *spleen* is enlarged. The vomiting and epigastric pain may be so pronounced as to mask the pulmonary symptoms. The occasional presence of jaundice has caused pneumonia to be mistaken for hepatitis, congestion of the liver, and even for gallstones. I saw a case of pneumonia, said to be appendicitis and peritonitis because of the characteristic pain, colic, and vomiting, followed by great abdominal tenderness in the upper abdomen.

Blood. Leucocytosis is a marked accompaniment of pneumonia, especially in cases ending favorably. The white cells may be increased from 12,000 to 40,000. An increase in the polymorphonuclear cells is always present. They fall with the crisis, or probably a day after the termination of the fever. In malignant forms there may be no leucocytosis. Increase in the fibrin network, causing the "buffy coat" of older writers, is commonly seen.

Cutaneous Symptoms. Herpes on the lips, the nose, or the genitals is of common occurrence. Sweating occurs with the crisis, or if heart-failure is imminent.

Urine. The urine is scanty and high-colored, and may contain a small amount of albumin. In some cases the chlorides are found to be absent. This is determined by acidulating the urine with a drop or two of nitric acid, and then adding one or two drops of a 10 per cent. solution of silver nitrate. If chlorides are present, a heavy white cloud of silver chloride is thrown down. The chlorides are not invariably absent, or even diminished in pneumonia, hence their reappearance, which is said to indicate beginning convalescence, loses its value as a prognostic sign.

Physical Signs. (See Diseases of the Lungs, Plate XXI.) **Consolidation.** This consists in a diminution of the amount of air and an increase of solid contents. On *inspection*, diminished movement. If extensive consolidation, enlargement of the affected side. On *palpation*,

inspection confirmed and increased vocal fremitus discovered. Both are more marked at the height of consolidation. *Percussion.* In the first stage, impaired resonance or Skodaic resonance. In the stage of hepatization, dulness or flatness, but without any wooden quality or marked resistance. *Auscultation.* In the early stage, that of congestion, the respiratory murmur is suppressed and crepitant râles are heard at the end of inspiration. On full inspiration or after cough a bronchovesicular respiration is brought out. When consolidation has taken place, the respiratory murmur is bronchial. Râles, if present, are moist subcrepitant râles due to associated bronchitis, or a few crepitant râles may persist, and a friction-sound be heard. When resolution sets in, the crepitant râle reappears, quickly followed by moist subcrepitant râles, heard both on inspiration and expiration, while dulness gradually yields to impaired resonance. The respiration loses its bronchial character and again acquires a vesicular element before becoming completely normal. It may be a week or two, or many months, even in uncomplicated cases, before the percussion-note becomes perfectly clear and râles wholly disappear.

The physical signs are modified by the intensity of the inflammation in the lung structure and by the pleural complications. In *massive pneumonia*, for instance, the auscultatory signs are absent. On percussion, the lung is absolutely flat. There is no fremitus or tubular breathing. The physical signs resemble those of pleurisy with effusion. In *central pneumonia* the physical signs may be delayed until the third or fourth day. A few râles or feeble breath-sounds over a small area may be the only indication of a possible lung process. In the *aged* the physical signs are obscure. In patients with laryngeal disease or marked obstruction in the nasopharynx the physical signs may be indefinite. Bronchial breathing may not be heard unless the patient takes a full breath or coughs. In this class of cases, as well as in those with feeble respiratory movement, as the aged, the weak, and in those suffering from some other disease, as tuberculosis, the physical signs are not made out because of the deficiency of respiratory movements. The indefinite character of the physical signs makes the diagnosis all the more difficult, because it is in this class of subjects that the general symptoms of infection are very slight. Increased respiration may be the most suggestive sign. Slight elevation of the temperature and more or less stupor may be the only other clinical symptoms.

Duration and Course. The duration of the disease is from one to two weeks. It may subside by crisis on the third, fifth, seventh, or ninth day, or gradually by lysis. Crisis is marked by a profuse sweat, a copious discharge of limpid urine, or sometimes by a few loose movements of the bowels, accompanying a fall of temperature to or below normal. Instead of clearing up, the pneumonia may progress to suppuration, abscess, or gangrene. These conditions can be made out by the character and range of temperature, the general condition of the patient, the sputum, and the physical signs. Termination in abscess or gangrene is rare. In cases proceeding to a fatal issue the strength fails, respiration becomes more labored, and expectoration increasingly difficult. The number of respirations often diminishes, but the pulse continues frequent and often

becomes small and irregular. Physical examination shows diffuse bronchitis with œdema. The heart's action is irregular and rapid. The sounds are weak and feeble; the first becomes short and snappy like the second, and later both are weak or indistinct. Death may occur abruptly from convulsion, or more frequently from asphyxia due to œdema of the lungs, which in turn sets in on account of weakness of the heart or the development of heart-clot from cardiac asystole.

Varieties. Migratory Pneumonia. Sometimes, with the reappearance of abundant râles and increased expectoration, the fever continues high, or, if the temperature has fallen to normal, again rises, the patient is disinclined to take food, has a dry, brown tongue, and is often delirious. In such cases the pneumonia is probably extending in the lung already involved or has attacked the other lung.

Typhoid pneumonia is an unfortunate name for an adynamic form of the disease with typhoid symptoms. If it arises in the course of or complicates low fevers, it is usually of the typhoid type: but it occurs also in those much exhausted, in depraved health, or exposed to unhygienic surroundings. It is found also in cases of septicæmia, in Bright's disease, and in drunkards. It is a type occurring in the negroes in the southern part of the United States.

The characteristic features of this form of pneumonia are the great physical prostration and the weak heart action. The fever is high, the respiration and pulse frequent, and delirium and vomiting are more common than in the ordinary form. The skin sometimes has a dusky hue; the tongue is heavily coated, or may be dry and brown, and sordes collect on the teeth. The sputa may be rusty, and sometimes pure blood is expectorated. The disease may rapidly prove fatal or may linger for a long time, the patient only gradually coming out of a low typhoid state. It is always dangerous.

Bilious pneumonia is the name given to a type of pneumonia occurring in persons who are already suffering from malarial poisoning. The initial chill lasts longer, and the pain in the side, from coincident pleurisy, is more marked than in ordinary pneumonia. The fever is more remittent, and jaundice and vomiting are present.

Pneumonia in infants is characterized by nervous symptoms. Repeated convulsions and active delirium may be most pronounced, followed by torpor and coma. There is no sputum and but little cough. The apex of the lung is affected.

Pneumonia in the aged is characterized by latency of symptoms. There is but little cough and expectoration. A tendency to the typhoid state, however, is pronounced. The physical signs are obscure.

Pneumonia in alcoholic subjects also develops insidiously and may be masked by the symptoms of delirium tremens. The temperature may be the only indication of infection, as there is neither pain, cough, expectoration, nor dyspnœa.

Pneumonia with other Infections. *Staphylococcus* and *Streptococcus pyogenes*, the colon bacillus, and *Bacillus pneumoniæ* (Friedländer) are often found with the pneumococcus, and may predominate, inducing a *mixed infection*. The micro-organisms which cause diphtheria, typhoid

fever, influenza, and the plague may cause a condition which resembles that of lobar pneumonia in the extent of the consolidation. *Micrococcus lanceolatus* is found in increased numbers in the sputum of these cases. There is not, however, the same intensity of pulmonary symptoms. The respirations are not so hurried. The physical signs, while extensive, are obscure, and indicate rather a heavy lung (congested) than one greatly consolidated. There are impaired resonance, feeble breathing, and a few râles in a large number of cases.

It is this form of lobar pneumonia which is difficult to distinguish from bronchopneumonia or catarrhal pneumonia—an infection which usually begins in the upper air-passages. This form of local infection is considered in the chapter on Diseases of the Lungs.

Diagnosis. The diagnosis is based upon the aggregation of special symptoms. The mode of onset, the chill, the course of the fever, the pain in the chest, the cough, the peculiar expectoration, the dyspnoea, the abnormal pulse-respiration ratio, the peculiar character of breathing, the physical signs, and leucocytosis are the phenomena of the symptom-complex. It must be remembered that in children, in the aged, in drunkards, in cases of chronic disease, the type is different. In drunkards cerebral symptoms are more marked. In children the cerebral symptoms are more prominent, the expectoration often absent. In the aged the cough, the expectoration, and the fever are not pronounced; the former may be absent; the onset is insidious. The same onset and course occur in wasting diseases, as cancer, phthisis, Bright's disease, diabetes, and organic heart disease. In this class of cases a small patch of pneumonia, difficult to determine on physical examination, may be attended by the gravest general symptoms. In all of the above cases, if there is *fever* without cause, although no pulmonary symptoms are present, the lungs must be examined repeatedly. In many such cases the physical signs are obscured because respiratory action is enfeebled by the primary condition.

Pneumonia must be distinguished from other acute inflammatory affections of the lung and pleura and from acute tuberculo-pneumonic phthisis. The evidence for each is considered in the respective sections. The presence of leucocytosis serves to distinguish it from acute tuberculosis and from typhoid fever, meningitis, and influenza. To distinguish pneumonia from pleurisy with effusion, the aspirator may be used.

Bacteriological Diagnosis. Staining and microscopical examination of the sputum reveal the characteristic micro-organism. Care must be taken to secure the sputum from the lung. By inoculation of rabbits with the sputum the disease is readily reproduced. The organism is not readily, although it frequently has been, found in the blood. (See the Sputum.)

In certain cases pneumonia may be distinguished from *cerebrospinal meningitis* by the results of spinal puncture alone; from *acute tuberculous pneumonia* by the examination of the sputum. The diagnosis in the latter instance may be postponed, as tubercle bacilli are sometimes not found until the tenth or twelfth day. (See Tuberculosis.) Typhoid fever sometimes resembles pneumonia, and must be distinguished after the first week by the results of serum diagnosis.

Pneumococcus Septicæmia.

The account we have just given of pneumonia represents but one phase of the pneumococcus infection. This infection may be attended by very grave symptoms, especially those of a toxic nature, with but little if any involvement of the lung tissue. It is well known that we may see the chill, fever, rapid pulse, and hurried respiration, with but little evidence of consolidation in the lung, but with nervous symptoms paramount. Delirium, stupor, coma, with the phenomena of the ataxic or the typhoid state, may prevail. (See pages 358 and 359.) In the ataxic state the symptoms resemble those of mania. In the typhoid form they are not unlike those of uræmia. In either instance death ensues in coma or from heart-failure with its attending symptoms. Preceding the cardiac failure the urine is diminished in amount and the secretions generally suppressed. The pneumococcus may be recovered from the blood, as has been done fairly frequent.

In other forms of this infection the localization of the process is in the pleura, as in empyema, in the pericardium, in the endocardium, and in the cerebral meninges. Pneumococcus inflammation of these structures is very common. It may develop at the same time that the lungs are affected, independently of the process in the lungs, or subsequent to it. These forms will be considered in a discussion of the various local inflammations just referred to.

It is important to remember that in pleural, pericardial, and cerebro-spinal infections the nature of the infection can be determined by *aspiration* and bacteriological examination of the fluid removed from the infected serous cavity. The pneumococcus infection can be positively diagnosed in this manner.

These complications, which occur in the course of the disease, modify the clinical picture and obscure the diagnosis.

Tuberculosis.

The infection discussed in this section prevails to a greater degree than that of all the others combined. In some forms, as pointed out in the clinical description, *fever* is one of the gravest symptoms. In other forms the febrile process may not be pronounced. It must be remembered that the fever may be due to the specific micro-organism or its toxin, or it may be due to a mixed infection. Staphylococcus and streptococcus infections are common attendants upon the tuberculous infection. This secondary infection may disappear or may become the most prominent infection. In many instances a terminal infection ensues, and is the cause of death. Infection by the pneumococcus is the most common of these terminal infections. (See page 374.)

Tuberculosis is an infectious disease, the course of which may be acute or chronic. It is caused by *Bacillus tuberculosis*. This micro-organism sets up a specific inflammation characterized by the development of nodules or tubercles, or by a diffuse growth of tuberculous tissue. Either anatomical product may undergo caseation or sclerosis, and in either instance ulceration or calcareous degeneration may set in.

Invasion of the body by the micro-organism may give rise to general infection, with an eruption of miliary tubercles in most of the organs and structures of the body, or to a local infection. General tuberculosis is acute; local tuberculosis may be acute or chronic. In acute tuberculosis the serous membranes, the lungs, liver, kidneys, lymphatic glands and spleen, the bone-marrow, and choroid coat of the eye may be invaded in whole or in part. In chronic tuberculosis the lymph-glands, the lungs, the serous membranes, the tissues and organs of the alimentary canal, the liver, the organs of the genito-urinary system, and the brain and cord are individually invaded.

Diagnosis. The diagnosis of any form of tuberculosis is aided by the determination of the chief factors in its etiology, when this is possible.

Bacteriological Diagnosis. *First.* The discovery of *Bacillus tuberculosis* in any inflammatory area, or any product of inflammation, as serum, blood, pus, or the secretion from any gland or mucous membrane invaded by the disease, establishes at once the diagnosis of this condition. The method of determining the presence of this micro-organism is fully detailed in the various descriptions of tuberculosis, in the discussion of local diseases, and in the accounts of the examination of the sputum, exudates, and transudates. *Inoculation* of inflammatory products, as of a gland or of a fluid which has been sedimented, is a positive mode of diagnosis. Guinea-pigs are selected for inoculation.

Second. As tuberculosis is an infectious disease, discovery of the *source* of the infection is an aid in the diagnosis. Infection takes place through the inhalation of the sputum or other secretions, which when dry float in the air. It implies in a measure more or less contact with individuals previously infected. In rare cases such contact is productive of the disease by means of direct contagion. The second source of infection is the food-supply. This may occur from the consumption of milk secured from a cow infected with tuberculosis. The eating of meat of tuberculous animals may possibly lead to infection. Direct inoculation is another but rarer source of infection, and usually occurs accidentally.

Third. It is possible that tuberculosis may be *inherited*. A more prominent ætiological factor, which aids in the diagnosis of the disease, is the presence of a certain type of structure which is a marked hereditary characteristic in families, on account of which feeble resistance is offered to the invasion of the tubercle bacillus. The phthisical and phthisinoid chest which belongs to this type has been described elsewhere, and the tuberculous and scrofulous states have been outlined. (See page 139 and Chapter XXXV., Part I.) These anatomical conditions, which are inherited, undoubtedly favor the development of tuberculosis.

It is a mistake to lay much stress in the diagnosis of tuberculosis upon the age or the occupation of the individual. Tuberculosis may occur at any age. It is true, however, that at certain periods of life the tubercles are distributed more commonly in one group of organs, while in other periods they affect another group. Lymphatic, joint-, and meningeal tuberculosis is most common in the first decade of life; the mesenteric glands are particularly subject to invasion at this period.

The diagnosis of tuberculosis, whether local or general, is further aided

by a complete knowledge of the phenomena that attend the entrance of the virus into the body and the mode of diffusion throughout the body after infection has taken place. The phenomena at the point of entrance of the micro-organism are nearly always distinct.

General Tuberculosis. The *general* invasion is associated with symptoms like those of specific fevers. The local secondary effects upon the tissues are always decided. It must be borne in mind that after the exposure, which may lead to infection, either an acute form of tuberculosis of a general character may be set up, with or without marked local symptoms, or acute local tuberculosis alone may arise.

Local Tuberculosis. In local tuberculosis the disease is confined to one organ or to the lymphatic glands and the organs in the lymphatic distribution, as the bronchial glands, which are primarily affected, and to the lungs. In these structures the entire process of nodular formation, caseation or sclerosis, ulceration or calcification, may take place. The disease remains primarily local. On the other hand, it may spread by continuity of structure through the lymphatics throughout the remainder of the organ affected, leading to its ultimate destruction and the death of the patient; or general infection of the system may take place from the primary local area. The primary seat of infection may be the lungs, the larynx, the alimentary tract, or the genito-urinary organs. Primary tuberculosis of the serous membranes, of the lymph-glands, of the bones and joints, may take place.

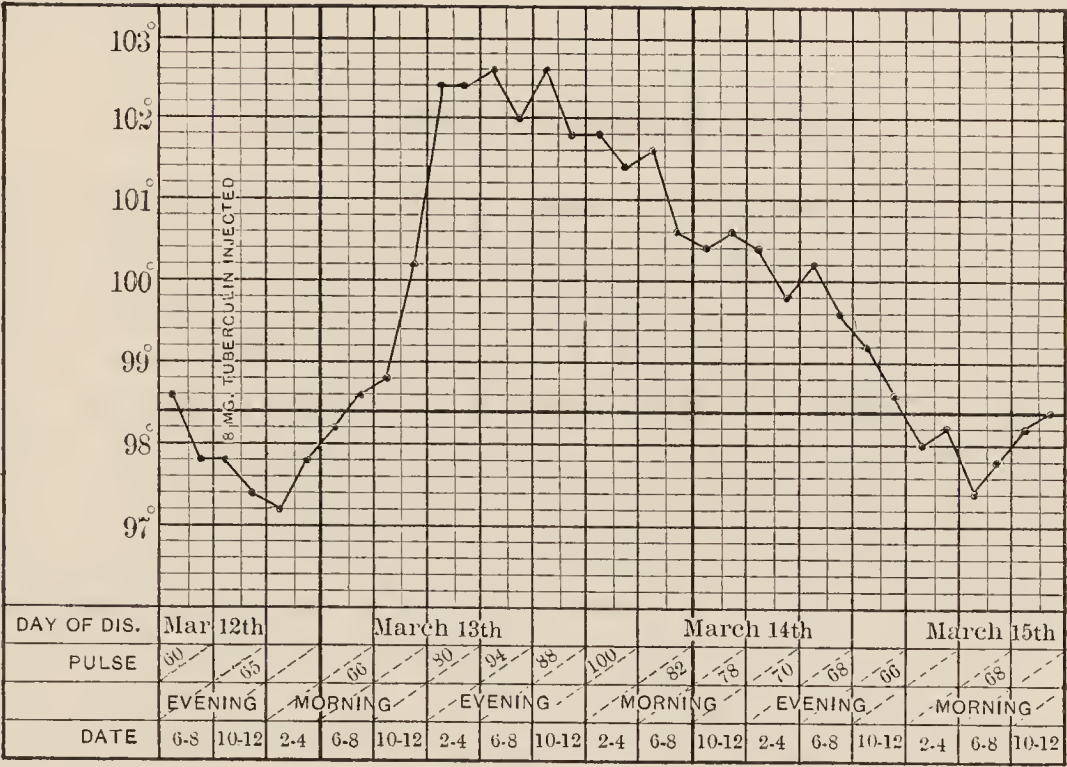
The symptomatology and diagnosis of the various forms of tuberculosis are detailed in the section devoted to the special diseases of the various organs of the body.

The Tuberculin Test. The physical signs and clinical symptoms may point to an inflammatory process in one of the many structures of the body which may be invaded by tubercle bacilli. On the other hand, failure in health, loss of weight, anæmia, and moderate fever may alone occur. The nature of the inflammatory process may be obscure. To determine more accurately whether the inflammation is tuberculous or not, or the "decline" due to tuberculosis, we can resort to the use of tuberculin. Since the researches of Koch, who introduced tuberculin as a remedy in tuberculosis, he himself as well as a number of other observers have employed this preparation to determine the presence of tuberculosis in the body. In this country Trudeau has been the earliest and most earnest exponent of this method of diagnosis. After the injection of tuberculin a group of phenomena follows, known as the tuberculin *reaction*, if tuberculosis exists anywhere in the body. It was thought the occurrence of this reaction was necessary to bring about a cure. As a therapeutic measure its value has not been upheld by experience. The invariable production of the reaction has led to its use as a diagnostic agent.

Phenomena of Reaction. About twelve hours after the injection of tuberculin the temperature rises rapidly. In the course of a few hours it has risen two or three degrees. This elevation of temperature is attended by malaise, pains in the head, back, and legs, and sometimes nausea or vomiting. The maximum temperature is maintained for two or three hours, and then a gradual decline to the normal takes place.

The normal temperature is reached in from twenty-four to thirty-six hours. The whole period of the reaction, from the time of the injection until the termination of the fever, is about forty-eight hours. With the fall of temperature to normal the constitutional symptoms subside. The accompanying chart (Fig. 311) shows the course of the fever in a typical reaction.

FIG. 311.



Typical reaction with tuberculin. (Original.)

Method. For twenty-four to forty-eight hours preceding the test the patient's temperature should be taken every two hours to determine the range at this period of the disease. The injection should be made at a time when the reaction can be observed—*i. e.*, during the period of normal or subnormal temperature if the temperature of the disease is intermittent. The hour of day selected to inject the tuberculin should be such that the reaction may be conveniently observed during the waking hours of the patient. Bedtime or the early morning hours are the most convenient.

The site of the injection is not important; usually the interscapular space is selected. The amount of tuberculin employed is of the greatest importance. The initial dose should never exceed 5 milligrammes, and it is better to use less than this, and an increasing quantity injected every second or third day. The maximum dose should not exceed 10 milligrammes. For children one-twentieth to one-tenth of a milligramme may be the initial dose. The crude tuberculin should be diluted with a 1 to 2 per cent. solution of carbolic acid at the time it is used.

At the point of injection a little redness and infiltration, with tenderness to the touch, are observed. This local reaction may also be seen at the site of former negative injections when the larger dose produces reaction. In pulmonary tuberculosis in which physical signs are obscure some auscultatory phenomena which were previously absent may be found during the period of a reaction. The test also enables one to detect tuberculosis in the pleura, pericardium, peritoneum, genito-urinary tract,

and lymphatic glands, the meninges, bones, and the skin. The test is of special value in cervical adenitis.

It must be remembered that a negative result with large doses of tuberculin is of more value than a positive one. In the former instance one can affirm that tuberculosis is absent, as well as that there is no old focus in any of the organs. It must also be remembered that the test should only be employed after all other means have failed to make a positive diagnosis.

Acute Miliary Tuberculosis.

This form of the infection has been spoken of elsewhere. (See Chapter VI., Part II.) Its course may resemble typhoid fever, septicæmia, or malignant endocarditis. It usually develops in the course of tuberculosis in some other organ of the body. The typhoid form has been described in the section indicated. It must not be forgotten that the diagnosis is rendered positive by the demonstration of the presence of tubercle bacilli in the blood, or of the occurrence of choroidal tubercles in the eye-ground. Another form is attended by marked pulmonary symptoms, which is seen in the bronchial pneumonia that occurs in children following measles and whooping-cough. (See Catarrhal Pneumonia.) Of the pulmonary symptoms, dyspnœa is the most prominent. Cyanosis is marked. The physical signs are not prominent, and may be those of bronchitis alone. Although there is impaired resonance at the base of the lungs, areas of hyper-resonance are observed above and anteriorly. Collapse of the lung may cause tubular breathing. The temperature rises to 102° or 103° F. An inverse type may be seen.

The diagnosis of acute tuberculosis is determined by the history of infection from extraneous sources or from local tuberculosis in some portion of the body, and by the presence of bacilli.

The following conditions should point to the possibility of chronic tuberculosis in some portion of the body: (1) emaciation, not otherwise explained; (2) chlorosis or anæmia; (3) weakness without cause; (4) fever—the temperature should be taken every two hours during night and day; (5) causeless sweats; (6) gastro-intestinal catarrh; (7) morning nausea; (8) signs of local inflammation in some organ of the body.

Influenza.

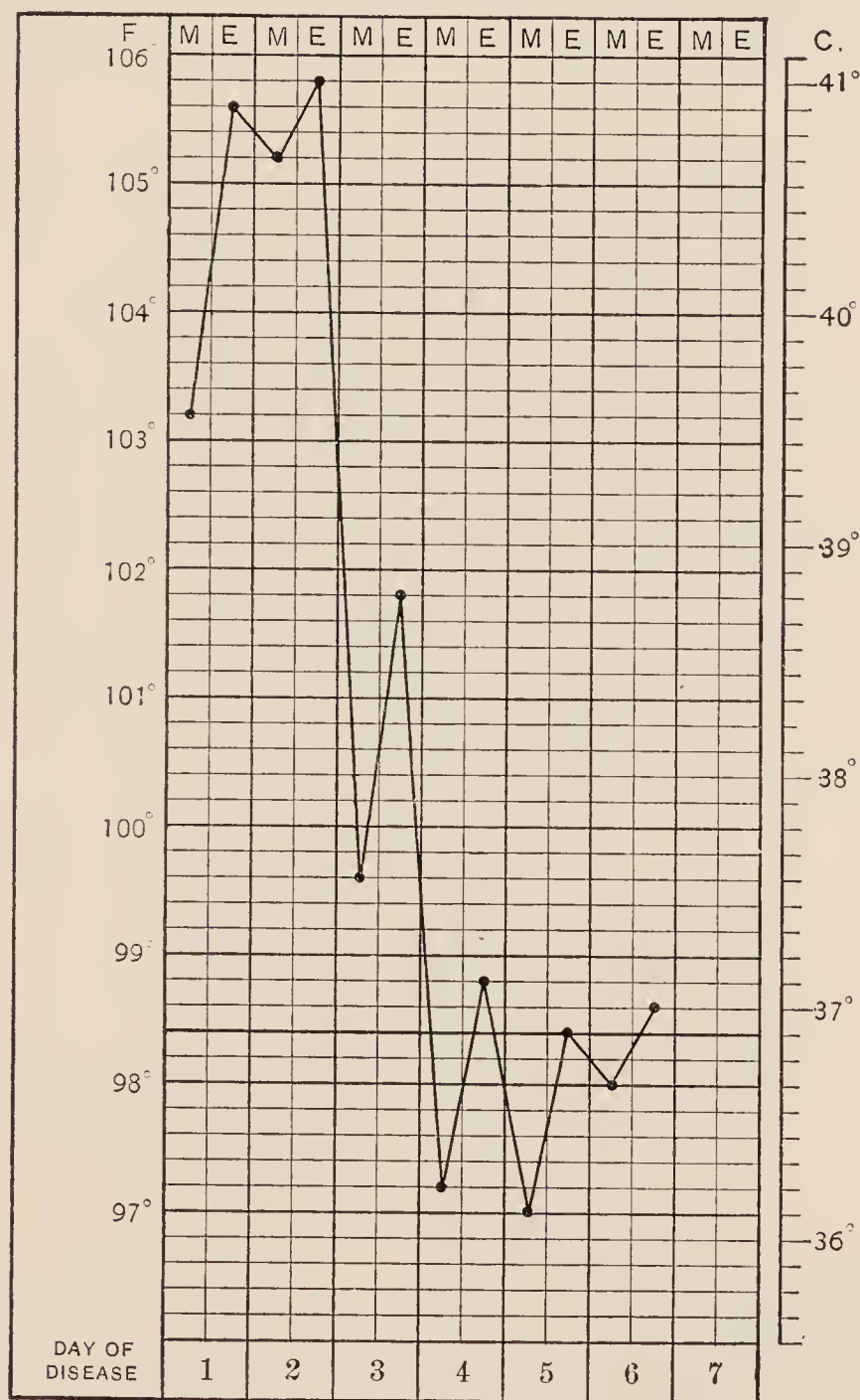
High temperature out of proportion to the local signs of inflammation in the lungs or other structures characterizes this infection. The *fever* may be continuous, remittent, or intermittent.

Influenza is a specific contagious febrile disease, occurring in widespread epidemics, having a very short period of incubation, and characterized by great prostration, marked nervous symptoms, and catarrhal inflammation of the respiratory or gastro-intestinal tracts, or both. There is great liability to relapse, and to complications, which are generally pulmonary or nervous.

The disease generally begins with the ordinary symptoms of coryza;

but the headache over the eyes and root of the nose is more severe, and may be so agonizing as to mask all other symptoms. The lacrymation, rhinitis, and tormenting cough are all usually worse than in ordinary coryza. Physical weakness, weariness, and depression of spirits are almost invariably present, and they sometimes reach an extraordinary degree. Fever is usually moderate (100° to 102° F.), but may be 104° to

FIG. 312.



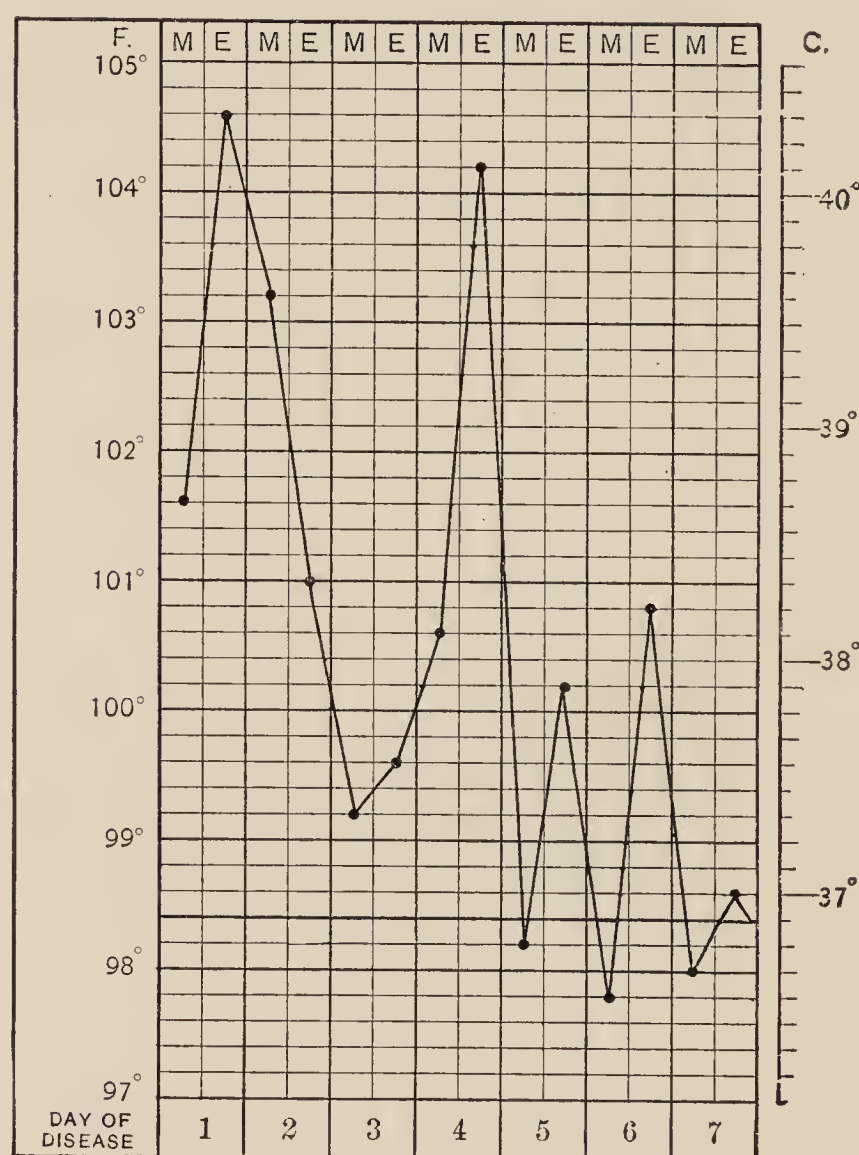
Temperature in influenza—interrupted crisis. (WILSON.)

105° F. for several days, and then gradually subside. It may terminate by crisis (Fig. 312), or may assume an intermittent or remittent type (Figs. 313 and 314). In ordinary cases the patient seeks relief first for the headache, severe aching pain in back and limbs, and extreme weakness; if these are relieved, he is apt to complain most of incessant racking cough, often due more to a tracheitis than to bronchitis. Nausea and vomiting are not uncommon, especially in the morning, at which time also the patient frequently feels worse than he does later in the day. Sleep is broken and restless, and may be accompanied by drenching perspirations. Severe neuralgic pains are common. In some cases the disease attacks the stomach and bowels especially, and vomiting and diarrhoea

are the prominent symptoms. In other cases the predominant symptoms are nervous, and great pain with prostration masks any catarrhal symptoms that may be present. Torpor and delirium may be present. Sometimes a prolonged and severe attack of asthma marks infection in susceptible persons.

The duration of the disease is from a few days to a few weeks. Convalescence is remarkably tedious, and is characterized by persistent weakness. Sweats are often annoying during this time. The heart often continues for some time to beat too frequently and to be easily excited by exertion. Relapses are common.

FIG. 313.



Influenza—intermittent type. (WILSON.)

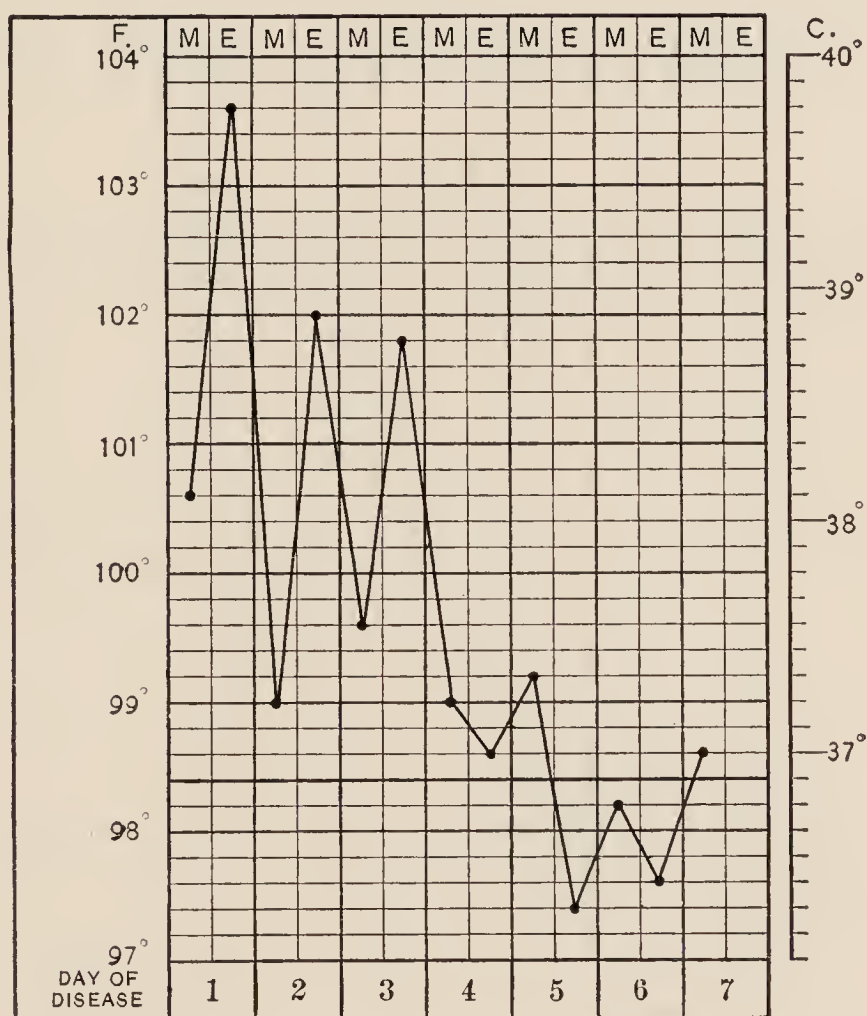
Diagnosis. Bacteriological Diagnosis. This is possible when the characteristic bacilli are detected by the means described in the section on Sputum. Influenza in the great majority of cases is easily recognized. In certain cases, however, it is to be differentiated from *pneumonia*, *typhoid fever*, and *cerebrospinal meningitis*.

Cases in which the disease sets in with high fever and marked chest symptoms are very apt to be mistaken for *pneumonia*; but the headache and prostration are more intense, while the respiration is not so frequent. Sweats are common, and albumin and casts in the urine are by no means rare. Physical exploration shows that both lungs are involved, though often not to the same degree. Resonance is impaired, and auscultation

shows moist crepitant and subcrepitant râles, which seem to be due to an œdematous condition of the lung tissue associated with a diffused bronchitis. A true lobar pneumonia is rarely present even as a complication.

If diarrhœa is one of the symptoms, *typhoid fever* has to be excluded. This is extremely difficult in the first two or three days. As a rule, headache, backache, nausea, and sleeplessness are at this time greater in influenza, the spleen is not so much if at all enlarged, the diarrhœa can be checked, and tenderness and pain in the right iliac fossa are absent.

FIG. 314.



Influenza—remittent type. (WILSON.)

Influenza can be distinguished from *cerebrospinal meningitis* by noting the fact that it begins with coryza, whereas the latter often sets in with chill, vomiting, and faintness; the headache in the former is usually frontal, in the latter occipital, and accompanied by stiffness of the back of the neck. Further, in cerebrospinal meningitis there are often swellings of the joints, and delirium alternating with coma, and in young subjects convulsions are common.

Finally, it may be said that the pronounced diagnostic feature is the preponderance of general symptoms over local inflammations. The occurrence of undue exhaustion, extreme general neuralgias and myalgias, high fever, and profuse sweats, without intense catarrh or inflammation to account for or to co-ordinate with them, is of the highest diagnostic significance. The presence of an epidemic, the contagious nature of the affection, the sudden onset, and the bacteriological diagnosis, all point to influenza.

Cerebrospinal Fever.

Cerebrospinal fever, also known as spotted fever, is an acute, specific, infectious, and mildly contagious disease, endemic and epidemic, characterized by evidences of *systemic infection*, and generally also by symptoms depending upon inflammation of the cerebral and spinal meninges—particularly intense pain in the back and head, hyperæsthesia, retraction of head and neck, delirium, coma, convulsions, and vomiting.

It is most common in cold weather, and in children under fifteen years of age. None of the epidemics show a continuous extension. The period of incubation is unknown, but is probably short. It is free from symptoms. The invasion of the disease is abrupt, although in some instances the patient may complain of rheumatoid pains in the limbs or a

FIG. 315.



Cerebrospinal meningitis, showing irregularity of pulse and temperature. (COUNCILMAN.)

joint, and headache and weakness. Usually the first symptom is a severe chill, which may awaken the patient from sleep. In other cases the initial symptom is a convulsion. Then quickly follow repeated vomiting, intense headache, sometimes accompanied by backache, retraction of the head, delirium, and extreme prostration.

In this infection more than all others the course of the *temperature* is without diagnostic significance unless it be that the absence of a characteristic course is significant. Its extraordinary irregularity is most striking when a large number of charts are examined. The fever may have the course and exacerbation of a typhoid temperature, but as a rule it is more like that of tuberculosis. It is often of very short duration, followed by a prolonged subnormal temperature. It may be high from the onset of the disease, or remain below 100° for several days, and then suddenly rise to a great height. Remissions and exacerbations may attend many of the cases. The most marked feature, apart from the irregularity of the temperature, is the inequality between the pulse and the temperature. In some instances the pulse is rapid, and the tempera-

ture is normal or subnormal, while later in the disease the pulse may be slow when the temperature rises to a considerable height. The temperature then may be said to be intermittent, remittent, or continuous; it may be intermittent at one period, continuous at another; it may be afebrile at one period and continuous at another.

Symptoms. The face is pale and livid, expressive of suffering, and the patient may toss from one side of the bed to the other, begging relief for his headache. Simple stiffness of the muscles of the neck may prevail. The pain in the head may be occipital or frontal. The pain in the back becomes more severe, and root-pains dart in all directions, but especially into the limbs or joints, which may be swollen and tender to the touch; in fact, the whole skin is hyperæsthetic and the reflexes are increased. The spinal muscles become rigid and the head is often retracted. Less frequently trismus occurs and the back is arched. Delirium is common at night. It may develop very early or appear at a late period of the disease. It is sometimes violent or low and muttering. It is often of a sportive type, the patient making absurd remarks, cracking jokes, or singing snatches of a comic song. Delirium may alternate with tonic or clonic convulsions and with stupor. The appetite is poor, the bowels constipated. A remission may occur on the third day, with temporary improvement of the symptoms.

As the attack progresses there may be strabismus, which is usually divergent, inequality of the pupils, nystagmus, ptosis, and optic neuritis. Vertigo, tinnitus, anosmia, and photophobia are common. Hyperæsthesia and delirium persist. Facial paralysis, a monoplégia, a hemiplegia, or a paraplegia may occur. The pulse becomes more frequent and the fever continues. In favorable cases improvement now begins, the headache and root-pains abating, and delirium and spasms becoming less frequent. In unfavorable cases the convulsions may become more severe and end in fatal coma, or the patient may sink into a typhoid condition, with nephritis as a complication. Coma may come on in the beginning and continue until death.

Kernig's Sign (Kernig, 1884; Netter, 1898). This sign is of value only in the diagnosis of meningitis in general. It is determined by placing the child in the dorsal position, with the legs relaxed and fully extended at the knees. When the child is raised to a sitting posture, the knees are flexed, and can not be extended on account of contracture of the posterior muscles of the thigh. In adults, if the patient is propped up, or seated on the side of the bed, and an attempt made to extend the leg on the thigh, there is contraction of the flexors. The test can be equally well performed by flexing the thigh on the abdomen until it makes a right angle. When an attempt is made to extend the leg, it will be found that the limb can not be fully extended if meningitis is present. "It is not always present in children under two years of age." (Packard.)

The *skin eruptions*, which explain the name "spotted fever," are not always present and exhibit no constant character. Herpes and petechiæ are the most frequent; in other cases the eruption is a purplish mottling, or is macular, or the eruption resembles that of measles. Herpes is most common on the nose and mouth, then on the cheek, forehead, eyes,

and ears. The blood shows a leucocytosis, the increase being due to the polynuclear leucocytes.

In the **malignant (fulminating) form** of the disease death occurs in a few hours, or in two or three days. Such cases are apt to arise early in an epidemic. The patient has a violent chill; delirium occurs early; the headache is less intense, or at any rate gives way rapidly to stupor and coma. The pulse is frequent and feeble; there may be no rise of temperature, the skin being cool, clammy, and cyanotic. Local or general convulsions may occur. The eruption may be purpuric, and even ecchymoses may occur. The urine is scanty and contains albumin and casts.

Mild cases usually occur late in epidemics, and are characterized by severe aching in the head, back, and limbs, nausea, vomiting, vertigo, and prostration. They closely resemble the nervous type of influenza, and could easily escape recognition except during an epidemic.

An abortive form, ending in recovery in two or three days, and an intermittent form, with exacerbations on alternate days, have been described.

The *duration* of the disease is from a few hours to two or three months. In ordinary favorable cases there is decided improvement toward the end of the first week, and convalescence is established in two weeks. It may become chronic and last for weeks, or, as already stated, may be fatal in a few hours. Relapses are common in some epidemics.

The most frequent *complications* are those involving the lungs and heart, particularly pneumonia and endocarditis or pericarditis. Pneumonia often occurs so early that it is difficult to decide whether it is primary with marked nervous symptoms, or is only a complication of the cerebrospinal fever. Nephritis also occurs.

The most frequent *sequels* are deafness, blindness, headache, and local palsies.

Diagnosis. The diagnosis in the presence of an epidemic is not difficult, although an absolute diagnosis can only be made by *lumbar puncture*. The fluid withdrawn is more or less cloudy if the patient has meningitis. If it is the epidemic form, microscopical examination of stained cover-slips and cultures will expose the diplococcus. In some cases fluid can not be secured, either because the spinal canal is filled with membrane or the fluid is retained in the lateral ventricles.

The fluid is turbid early in the disease; in some cases a purulent sediment forms in the bottom of the test-tube at once. In others the fluid is simply turbid, and after standing contains considerable fibrin and many cells. The fluid secured at the first puncture may be more turbid than that secured later, although the symptoms may be more severe than at first. If the acute symptoms subside, the fluid may be clear and no cells may be found. In the intermittent cases the fluid may be clear during the interval that the patient is without symptoms. In chronic cases there may be no turbidity. The *cells* in the spinal fluid are chiefly polymorphonuclear leucocytes—"pus-corpuscles." Small lymphoid cells and large endothelial cells may be present. The latter are phagocytic, and have large oval or round nuclei. They may contain leucocytes and blood-corpuscles. In the pus-corpuscles or leucocytes the

PLATE XVIII.

Fig. 1.



Fig. 2.



Cerebro-Spinal Meningitis. (Councilman.)

Fig. 1. Forty-eight hour culture of *diplococcus intracellularis* on Loeffler's blood-serum mixture.

Fig. 2. Abundant growth in twenty-four hour culture on fresh blood-serum. The colonies are minute, very numerous, and somewhat resemble similar cultures of the pneumococcus.

diplococci are found; they are rarely found outside of the cells. Late in the disease the pus-corpuscles do not stain sharply and are degenerated. In chronic cases the fluid contains a few pus-corpuscles which are smaller than usual and similar to lymphoid cells.

Bacteriological Diagnosis. The diagnosis of this form of meningitis can often be made by staining, and by microscopical examination of products from the nasopharynx removed from the nares with a swab. The disease is due to *Diplococcus intracellularis*, which appears in the diplococcus form as two hemispheres the size of the ordinary micrococcus. It stains with the ordinary stains for bacteria. It is decolorized by the Gram method. The staining is sometimes irregular, some of the diplococci being brightly stained, others faintly. There is some variation in the size of the organisms. Both the variation in size and staining are apparently due to degeneration. The two cocci are sharply separated usually, though sometimes they seem to be united. (Figs. 316 and 317.)

FIG. 316.

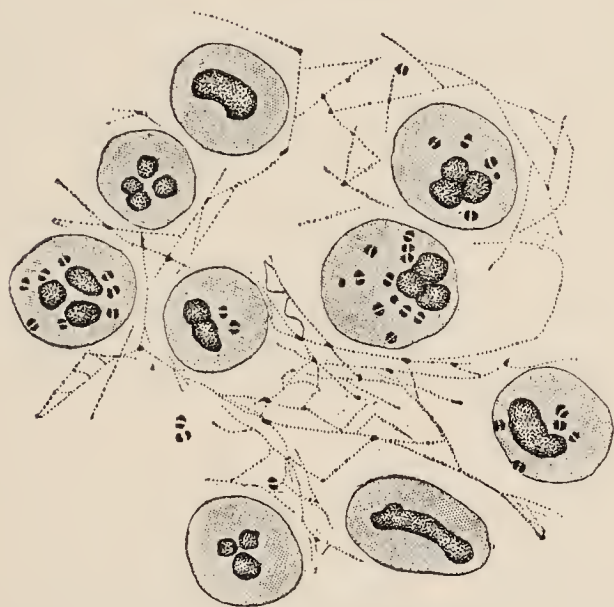


FIG. 317.

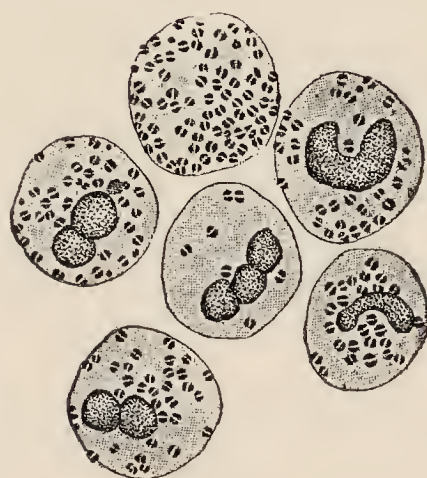


FIG. 316.—Pus-cells containing diplococci from the meninges. A few diplococci are in the exudate outside of the pus-cells. Between the pus-cells there are delicate fibrillæ of fibrin. The drawing is an accurate representation of a group of cells in the field of the microscope. (COUNCILMAN.)

FIG. 317.—Pus-cells from an alveolus of the lung in a case of diplococcus pneumonia. The cells are swollen and contain immense numbers of diplococci. Both figures from stained cover-slips.

CULTURES. Cultures should be made at the time of puncture. In the majority of cases a growth of the diplococcus is found, although rarely even in acute cases they may not grow. In chronic cases a growth is only rarely obtained. (Plate XVIII.) The micro-organism has been recovered from the blood, and from the fluid of an inflamed joint as well as from the spinal fluid.¹

The epidemic form of meningitis must be distinguished from pneumococcus meningitis, tuberculous meningitis, and streptococcus meningitis. In the pneumococcic form the symptoms are comparatively slight and are usually preceded by pneumonia. In the streptococcic form the clinical history is like that of ordinary forms of meningitis. The evidence of an infection elsewhere is usually present. Tuberculous meningitis is recognized by the methods employed to detect tuberculosis elsewhere in a patient suffering from the usual symptoms of cerebrospinal meningitis.

¹ Osler, Philadelphia Medical Journal, 1899.

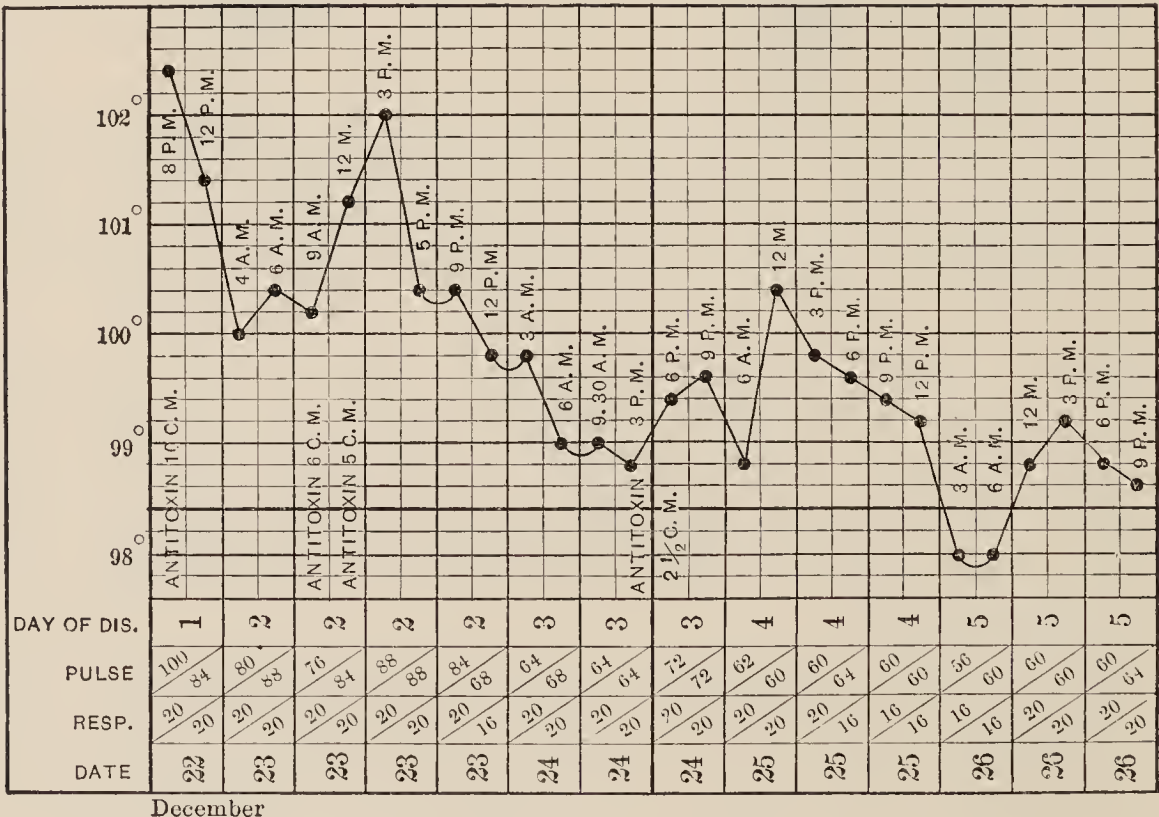
The most positive method of distinction of the various forms is by lumbar puncture. (See Chapter XLV., Part I.)

Diphtheria.

Diphtheria is an acute, specific, infectious, and contagious disease, sporadic and epidemic, occurring especially in children from one to six years of age, and characterized by insidious or abrupt onset, with moderate fever; and the development upon the fauces or upon any abraded surface of a grayish-white false membrane, which has a tendency to extend, especially to the larynx. The subsequent phenomena are those of stenosis of the larynx, toxæmia, with or without superadded uræmia, or marked cardiac weakness; it is further characterized by the liability to paralysis as a sequel.

Diphtheria is contracted by inhaling the expired breath of a diphtheritic patient, or breathing air which has been contaminated by the clothing of the patient or the discharges from his nose or throat. It may also be transmitted directly, as when a fragment of membrane is ejected by coughing and infects the mouth or eye of the physician or attendant. Moreover, air contaminated by diphtheria is contained in the sewers of large cities while the disease is endemic, and may persist in damp cellars if they have once been infected. Hence, sewer-gas and cellar-air may carry the disease. There is reason also for believing that a similar disease affects birds, fowls, and cats at times, and by them may be transmitted to man. These facts must be borne in mind in making the diagnosis.

FIG. 318.



Diphtheria. (Original.)

The specific poison is the Klebs-Löffler bacillus and its toxin.

While children from one to six years of age are especially liable to it, no age is exempt—neither the new-born babe nor the very aged. One attack does not protect a person completely against a subsequent attack.

The temperature range is variable. The infection may be intense, and yet the temperature remain subnormal, especially if the *fever* is due to the toxin, and not, as is frequently the case, to a mixed infection.

The period of *incubation* varies from a few days to two weeks, or perhaps longer in exceptional cases. As a rule it is less than a week. It is shorter when the poison is virulent and when infection has been upon abraded surfaces.

The *onset* in mild cases is deceptively free from positive symptoms. The child is languid, perhaps slightly chilly, and has a little fever, with thirst, impaired appetite, and discomfort in swallowing. Unless the nature of the trouble is suspected, the child is not thought ill enough to be kept in-doors. The throat is slightly inflamed, especially about the tonsils. The child may protest that there is no pain on swallowing. In from twelve to twenty-four hours after the onset, sometimes later, a grayish pellicle will be found upon the tonsils, and the cervical glands will be swollen. In more severe cases the disease begins with chill or chilliness, followed by a rise in the temperature to 102° or 104° F., sore throat, and sometimes vomiting, though this is not so common as in scarlatina. Convulsions and delirium may occur if the fever be high or the case malignant, but they are not common. The disgust for food makes it difficult to nourish the patient. Headache, thirst, and aching in the back and limbs may be complained of. Prostration is often very pronounced from the beginning.

Objective Symptoms. The characteristic false membrane appears at first as a grayish pellicle upon one or both tonsils, and spreads thence to the soft palate and pharynx. The membrane soon becomes thicker and whitish in color; when fully developed, it appears like white or grayish-white parchment, not lying loosely upon the surface, but imbedded in the mucous membrane, the inflamed swollen edges of which rise above the false membrane, surrounding it "as the crystal of a watch is surrounded by the rim." (J. Lewis Smith.¹) As the membrane becomes older it may be brownish or even blackish in color if ferric chloride has been given. If it is forcibly torn from the underlying surface, hemorrhage is excited, and the membrane forms again. As the membrane loosens spontaneously there is often marked inflammatory reaction at the edges of the surrounding mucous membrane, and in the tonsils there may be decided sloughing, with a dark, gangrenous appearance.

The *temperature* usually falls on the second or third day, but this does not indicate either a favorable or an unfavorable end. A temperature slightly above normal is not uncommon in profound toxæmia.

Urine. Albumin is usually present early, and often tube-casts and renal epithelium also can be found. The submaxillary and cervical glands are swollen, and it may be difficult to open the mouth sufficiently to inspect the throat.

As pointed out by Buzzard and McDonnell, the patellar tendon reflexes are often abolished as early as the first day.

In *favorable cases* the membrane ceases to extend after three or four days; there is no extension to the larynx; the urine is free from albumin,

¹ Keating's Cyclopædia of Diseases of Children, 1889, vol. i., p. 606.

or only slightly albuminous ; and the pulse-rate is not more than 100 to 120 per minute and of good force.

In *unfavorable cases* the membrane shows a tendency to extend upward into the nasal fossæ, producing a thin, irritating, excoriating discharge from the nostrils, and rendering mouth-breathing necessary ; or it may extend to the ears through the Eustachian tube ; into the maxillary sinus ; or downward into the larynx, producing laryngeal stenosis. This is announced by hoarseness, with rapidly increasing difficulty in breathing. Inspiration is high-pitched, noisy, and difficult ; the patient brings all the accessory muscles of respiration into action, the alæ of the nose play, the ribs are sucked in, and still the child pants for breath. Every now and then a paroxysm of coughing produces cyanosis.

In other unfavorable cases the throat symptoms are not dangerous, but *uræmia* develops. The urine is scanty, contains a large amount of albumin, considerable blood, and numerous epithelial and granular casts. There are œdema of the feet and puffiness of the eyelids. There is apt to be repeated vomiting ; convulsions, followed by coma, and death may end the scene, or the patient may slowly be restored.

Heart. In still other cases the diphtheritic poison affects the heart. The pulse becomes feeble and very frequent, the first sound very faint ; acute dilatation of the right heart may occur. There may be faintness and a tendency to cyanosis on the slightest provocation, or attacks of sinking and faintness may come without warning ; in still other cases sudden exertion induces paralysis of the heart and death.

In some *malignant cases* the patient is overwhelmed by a large dose of the poison, and dies in from one to three days in collapse from acute toxæmia, without any special local symptoms to account for it. In others the false membrane extends rapidly over the fauces, pharynx, and nasal cavities to the larynx ; death occurs from early obstruction, or, if it is postponed, there is extensive sloughing, with death from secondary blood-poisoning or septic pneumonia.

In exceptional cases the membrane is primary in the nares or larynx, or develops upon some abraded surface, as a burn, or in the vagina of a puerperal woman. It may also attack the mucous membrane of the eye or the seat of a recent operation. Diphtheria also occurs as a complication of other diseases, particularly scarlet fever.

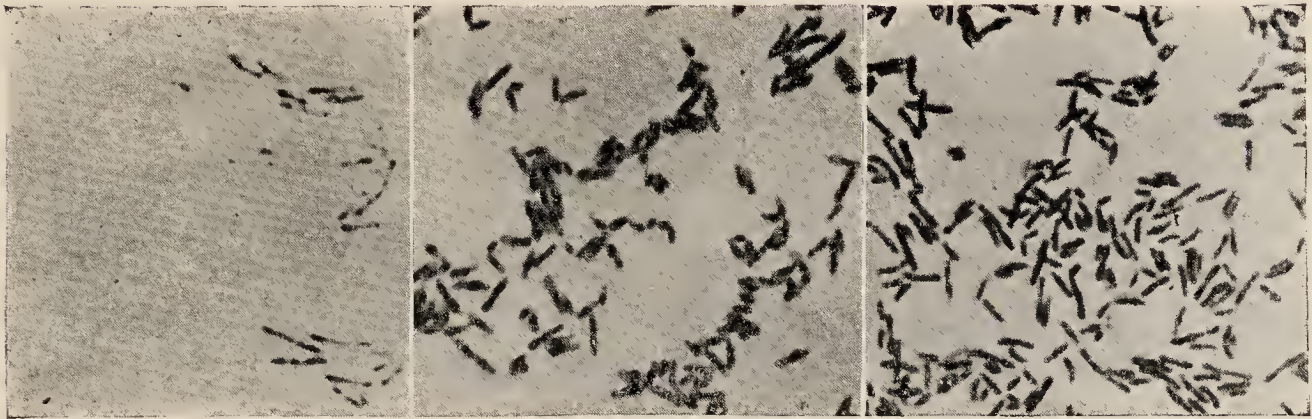
Sequelæ. The most frequent sequelæ are anæmia, albuminuria, and paralysis. The latter comes on in from one to two weeks after convalescence has set in, but it may appear much earlier, and in exceptional cases later. It may be marked simply by loss of the knee-jerk, which has been alluded to, or involve the palatal and pharyngeal muscles, causing nasal speech, difficulty in swallowing, and regurgitation of food through the nose, or there may be multiple peripheral neuritis.

Löffler's or the Klebs-Löffler Bacillus. This micro-organism is found in diphtheritic pseudomembranes, especially in the deeper portions, and has also been found in the blood.

Morphology. A bacillus $2\ \mu$ to $3\ \mu$ long by $0.5\ \mu$ to $0.8\ \mu$ broad, straight or slightly curved, with very many irregular forms. (See Fig. 319.)

The *pseudodiphtheritic bacillus* resembles the genuine in all respects, except that it is not pathogenic. It seems to be an attenuated variety of the former.

FIG. 319.

Diphtheria bacilli. $\times 1000$. (From LOOMIS and THOMPSON.)

Diagnosis. Diphtheria is distinguished from ordinary *pharyngitis* by the presence of membrane. From *follicular tonsillitis* by the projecting mouths of the follicles containing a creamy-white exudate. Later the exudate may cover the entire surface of each tonsil and be difficult to distinguish from false membrane. The points of distinction are that in the former the exudate lies upon the surface and can be brushed off without force and without leaving a bleeding surface; whereas in diphtheria the membrane is imbedded in the mucous membrane and can not be torn from it without some violence. A raised, red, inflammatory border of mucous membrane overhanging the patch is strongly suggestive of diphtheria. In tonsillitis there is no appearance of membrane upon the soft palate or pharynx. Furthermore, in tonsillitis the onset is attended with more fever and pain in swallowing than is true in simple tonsillar diphtheria. The existence of albuminuria and swelling of the cervical glands indicates diphtheria, and the absence of knee-jerk is an important but not a constant sign of diphtheria. The presence of the Klebs-Löffler bacilli in a culture from a suspected throat is proof of the existence of diphtheria.

Septicopyæmia.

The clinical course of this infection and the bacterial causes have been considered in Chapter XXXII., Part I. It will be recalled that its phenomena may attend a number of the infections described in this and in previous chapters. When occurring in the course of pneumonia, diphtheria, typhoid fever, etc., its origin is recognized by the methods discussed in the chapter referring to these infections. Septicopyæmia caused by pyogenic organisms, the so-called "cryptogenetic sepsis," is recognized by bacteriological examination of the blood; by an examination of the morbid secretions; or by an examination of the products of inflammation. Bacteriological examination of the blood has its limitations. Usually only late in the course of the disease and in the more intense infections can the bacteria be found. Examination of the pus from foci of suppuration in the bones (osteomyelitis), in the joints (pyæmia), in the serous cavities (empyema, pericarditis, peritonitis), in the lungs (see Sputum),

and in the genito-urinary tract (see Urine), will show the infective micro-organism.

The causal micro-organism is detected by cover-slip preparations and cultures. (See Chapter XLV., Part I.)

Glanders.

Glanders is an infectious constitutional disease, transmitted from horse to man, appearing in an acute and chronic form, and characterized by an eruption, ozæna, small tumors, ulcerations, cough, and death in coma or collapse in from one to four weeks in the acute form, or in three or four months in the chronic form. The symptoms in the latter resemble at times syphilis and at times tuberculosis.

The disease is rare in man. It may be acquired by direct inoculation of an open wound with the pus from a glanderous ulcer or nasal mucous membrane, or indirectly from infected straw or other material. The raw meat of a glandered animal also has infective power.

The *fever* which attends this infection is similar to that of the infective granulomata. In severe cases the symptoms are like those of an acute septicæmia.

In *acute glanders* the onset is marked by headache, slight fever, and pains in the limbs. If a wound has been infected, it becomes painful, swollen, and behaves like any poisoned wound. Sometimes a diffused redness, resembling erysipelas, spreads from the infected point. Fagge refers to a case in which the first complaint was of pain in the side and dyspnœa, so that acute pleuropneumonia was suspected. An eruption, consisting first of papules, which rapidly become flat vesicles and then pustules or bullæ, appears in the first day or two, or sometimes not for a week or even longer. (Fagge.) The bullæ or pustules rupture and give vent to a thin, purulent discharge. There may be hard, painful lumps in the muscles, with subsequent suppuration (farcy). Ozæna is not always present. It appears in the second or third week of the disease. The nasal discharge is mucopurulent, then purulent and fetid. The nose subsequently swells and becomes red and very painful. Ulcers and even necrosis of the septum are the lesions; a similar catarrhal condition may exist in the throat, eye, larynx, and mouth, accompanied at times by ulcers and false membrane. The patient gradually sinks into a septicæmic condition, with irregular fever, dry brown tongue, albuminuria, delirium, coma, and collapse.

The *duration* of the acute form is from one to four weeks. Only one in thirty-eight cases collected by Bollinger ended in recovery.

In the *chronic form* there are ulcers upon the hand, face, forehead, or elsewhere. In other cases the lesions are abscesses in the joints which are followed by persistent fistulæ. In still other cases there is pustular eruption. Ozæna may or may not exist. In still other cases the prominent symptoms are cough, bloody expectoration, hoarseness, fever, and emaciation. Bollinger reports seventeen recoveries in a total of thirty-four cases of chronic glanders.

Diagnosis. Acute glanders is distinguished from rheumatism by the

history of the case, the occupation of the patient, the existence of an open, irritable sore, and the fact that while the joints may be painful, they are rarely red and swollen as in rheumatism. Subsequently the appearance of pustules, bullæ, and ozæna makes the case clear. The same peculiar features serve to distinguish the disease from pyæmia, malignant pustule, and other infectious diseases. In a suspected case of chronic glanders a correct diagnosis might be arrived at by inoculating a mule or a horse with the nasal mucus or pus from a farcy.

Bacteriological Diagnosis. The specific germ is *Bacillus mallei*. This is a short, non-motile micro-organism resembling the tubercle bacillus. It is $2\ \mu$ to $3\ \mu$ long, and $0.3\ \mu$ to $0.4\ \mu$ broad, frequently having spores on the ends. It stains readily, although irregularly, with all the basic aniline dyes.

STRAUSS TEST. The examination for the bacillus is readily made by the method of Strauss. A portion of the suspected tissue or a culture from the lesions is inoculated into the peritoneal cavity of a male guinea-pig. If the case is one of glanders, the testicles begin to swell in about thirty hours, and an orchitis with abscess develops. The diagnostic sign is the tumefaction of the testicles.

MALLEIN TEST. Mallein is the filtered product of the growth of the bacillus on fluid media. It is allied to tuberculin. Injection in a suspected case produces a reaction similar in its course to the tuberculin reaction if the case is one of glanders.

Cholera.

An acute, specific, infectious disease, endemic in parts of India, but occurring in epidemics elsewhere, characterized by the outpouring into the stomach and bowels of large quantities of a serous fluid resembling rice-water, which fluid is usually vomited and discharged from the intestines. It is further characterized by an algid state of collapse and by painful muscular cramps.

The specific poison of cholera is believed to be the *comma bacillus* of Koch and its ptomain.

The native habitat of cholera is India, particularly the neighborhood of Calcutta; here it is endemic, and thence it is liable to spread in successive epidemic waves along the lines of travel by sea and land over the whole world. It is scarcely, if at all, contagious; the poison is contained in the vomitus and in the dejections, which contaminate the drinking-water, food, and clothing. The cholera bacillus preserves its vitality for long periods of time in water, especially if the water is slightly alkaline and contains vegetable matter, and in moist clothing, as rags.

The period of *incubation* is probably short in the majority of cases, lasting only a few days. Occasionally it is two weeks. There are usually no definite symptoms during this time, but there may be a sense of weakness, with loss of appetite and dyspeptic symptoms.

First Stage. The first stage, that of premonitory diarrhœa, is better regarded as the beginning of true cholera. It is characterized by profuse watery stools of a yellow or light-yellow color and alkaline reaction.

They are accompanied by a rumbling noise in the bowels, but are passed without pain. From six to a dozen of these passages occur in twenty-four hours. The patient feels faint and exhausted after them, and may suffer from nausea, but vomiting is not usual. In severe cases there may be cramps in the calves of the legs. The voice is faint and husky, thirst intense, the tongue white and moist. The temperature is normal or slightly depressed. This stage may last from two days to a week, depending upon treatment. In some cases it is absent, and the second stage sets in abruptly.

Second Stage. This usually comes on during the night. The patient is seized with vomiting, which is at first bilious, but rapidly loses all color and becomes like rice-water. The stools likewise resemble water in which meal has been stirred, or in which rice has been soaked—a semitransparent fluid, with particles of epithelium resembling rice floating in it. This fluid seems to well up and regurgitate rather than to be vomited from the stomach, and to gush in quantities of a quart or two from the anus. Sometimes vomiting and diarrhoea occur at once. The patient has unquenchable thirst, and is tortured with painful cramps of the toes, legs, belly, and diaphragm. As the discharges continue the patient becomes more and more exhausted; the nose is pinched and twisted, the eyes sunken, the lips bluish, and the whole body may shrink beyond recognizable proportions. The skin is cold and moist, the breath icy, and the temperature under the tongue is sometimes as low as 78° F. In the vagina and rectum it may be normal or slightly above normal. The patient, however, often has a sensation of heat. The urine is very scanty, containing albumin and sugar, or it may be suppressed. The pulse is very small and feeble, its rate 100 to 120. The mind is clear, but the patient is listless, answering questions in an extremely faint voice and with manifest effort.

Third Stage. From this collapse and algid condition the patient may slowly emerge, the skin becoming less cold, the cramps less severe.

A return of the secretion of urine is a hopeful sign. The reaction, however, may simply introduce a low typhoid condition, with fever, dry brown tongue, subsultus, low muttering delirium, and coma. In some cases serum is poured out into the stomach and intestines and is retained there. The patient while walking may be seized with dizziness, faintness, extreme prostration, and early collapse. In other cases the patient is smitten with profuse vomiting and purging, dying algid and collapsed in a few hours, no reaction appearing. In favorable cases the vomiting ceases, the stools become less frequent, and are tinged with bile and have a fecal odor. The urine increases in volume, while the albumin diminishes. Convalescence is protracted. Anæmia, great debility, feeble digestion, and sometimes obstinate diarrhoea delay complete recovery. Relapses are frequent. In other cases reaction brings improvement in the gastro-intestinal symptoms, but uræmia develops, death following in convulsions or coma.

The most frequent *complications* and *sequelæ* are eruptions, chiefly erythematous, ulcerations, bed-sores, and parotitis; and between the tenth and fifteenth days of convalescence a painful tetanic spasm may be seen of the flexor muscles of the hands, forearms, legs, and feet. (Stillé.)

Diagnosis. The chief points in the diagnosis from other affections are the knowledge of exposure to cholera; the character of the vomitus and dejecta, and the bacteriological findings; the cyanosis; the rapid development of collapse, with cold skin, icy breath, torturing cramps, and greatly shrunken visage and body.

Cholera morbus differs in that the stools are turbid with bile or fecal matter, or contain blood; they never present the rice-water appearance. Moreover, the passages are frequently preceded by colicky pains. Cyanosis and collapse are extremely rare. The stools do not contain the cholera bacillus.

Other forms of acute *toxic gastro-enteritis*, whether from ptomaine-poisoning or from corrosive poison, are to be distinguished by the history, the difference in the character of the stools, and the comparative mildness or absence of such symptoms as painful cramps in the legs, cyanosis, and collapse.

Bacteriological Diagnosis. Koch remarks:¹ "As cholera resembles in clinical symptoms cholera nostras, infantile cholera, certain forms of peritonitis, certain organic poisons, and poisoning by arsenic, it is important to attain some means of making a definite diagnosis." The bacteriological diagnosis is based upon the staining of smears, cultures, and inoculations.

SPIRILLUM CHOLERÆ ASIATICÆ (*the Comma Bacillus*). The comma bacillus of Koch is the specific causative agent of cholera. In a disease so widespread in times of epidemics and so fatal, it is of great importance to be able to recognize the bacterium that produces it.

MICROSCOPICAL EXAMINATION. The cholera bacillus is short, more or less bent, both shorter and thicker than the tubercle bacillus, and is generally shaped like a comma, but may form a half-circle, or two may be joined like an S. It has rounded ends, and is from $0.8\ \mu$ to $2\ \mu$ long and from $0.3\ \mu$ to $0.4\ \mu$ broad. The bacilli are often found placed end to end, and thus form a curve like a spiral. They are always present in the stools of cholera patients and sometimes in the vomitus. They are particularly abundant in the mucous floccules of the rice-water discharges, and can be obtained from the linen soiled by the same. Cover-slip preparations are made from these portions by placing a uniform film on the slip, drying it in the air, and then passing it through the flame of a Bunsen burner or spirit-lamp.

By Löffler's method a single flagellum is found on the rods. It stains with anilines, but slowly. An aqueous solution of fuchsin (Ziehl's red)

FIG. 320.



Cholera spirilla grown on moist linen. $\times 600$. Cultivated from the dejections after two days. (After KOCH.)

¹ Zeitschrift für Hygiene und Infektionskrankheiten, 1893, vol. xiv., No. 2.

is the best. In addition to the cholera bacilli, *Bacillus coli communis* and other intestinal bacteria are found. In thread-like strands of mucus the cholera bacilli form groups in which the bacilli all lie in the same direction. Koch holds that this mode of grouping is characteristic and diagnostic. He further holds that if *Bacilli coli* are in close proximity to numerous scattered bacteria resembling the cholera bacilli the case is one of Asiatic cholera.

The bacillus of cholera nostras and one found in cheese by Deneke resemble the comma bacillus in shape, though somewhat larger, but they have bacteriological peculiarities by which they can be differentiated.

Dysentery.

The fever which attends this infection is, from a clinical standpoint, the least characteristic symptom. It varies in part with the age of the patient. In the aged it is subnormal, normal, or moderate; in the young it is usually very high. It differs with the character of the infection; if a mixed infection prevails, the temperature is not unusual.

The term dysentery is applied to an inflammation of the intestinal tract, chiefly the colon, which is attended by the symptoms of intestinal catarrh in intense degree, with mucus and bloody discharges and the general symptoms of fever and prostration, followed by extreme exhaustion, and at times the occurrence of abscesses in the portal circulation, or of paralysis, arthritis, nephritis, or profound anæmia. It was formerly thought to be an epidemic, mildly contagious disease. Although of frequent occurrence sporadically, it is especially common in jails and institutions, in camps, or where people are crowded together, when at the same time hygienic conditions are most unfavorable. It usually occurs in the summer or fall, and is attributed to the drinking of impure water. A form most common in the tropics is called *tropical dysentery*. Recent investigations appear to show that catarrhal dysentery due to the above-mentioned circumstances, and "tropical" dysentery, which therefore is not necessarily confined to the tropics, are associated with inflammation and ulceration of the bowel, due to *Bacillus dysenterice*, the bacillus of Shiga, or *Amœba coli*.

Catarrhal Dysentery. Catarrhal dysentery may be limited to simple inflammation of the intestine or may be followed by ulceration. Its first symptoms are those of intestinal catarrh. There is indigestion, with loss of appetite, perhaps vomiting, and a slight diarrhœa. These symptoms may be the immediate effect of the inflammation. At the end of three or four days a chill may take place, showing the setting in of an infection. The diarrhœa is attended by pain, at first seated around the umbilicus; it then becomes marked in the course of the colon. The bowel movements are frequent, preceded by constant desire and attended by extreme tenesmus. The stools, which are first fecal and fluid, soon become scanty, and consist almost entirely of mucus and blood. The symptoms of local proctitis are severe; there is a sensation of a hot mass in the rectum. There may be strangury, and prolapse of the anus may ensue.

With the continuance of acute pain and frequent evacuations the skin becomes hot and dry; thirst, nausea, and occasionally vomiting occur. The temperature continues at about 103° F.; the pulse is rapid. The patient is weak and restless; the tongue is red and raw.

If the disease is severe from the start, or the course is unfavorable, the stools may contain pure blood, or they may be dark in color, and contain shreds of membrane. Pain and tenesmus disappear, and the evacuations become constant or involuntary. Restlessness is aggravated; the extremities become cold; mild delirium sets in. The tossing and restlessness are characteristic, and are attended by sighing and some dyspnoea. The pulse is rapid and feeble; the heart-sounds are weakened; the tongue becomes dry and brown, the mouth is parched, and thirst is intense; ulcers develop in the mouth and sordes collect around the teeth. The delirium increases to stupor, and from that to coma. The urine, at first high-colored and scanty, becomes bloody, and contains albumin and casts. Although the fever continues during this stage, the extremities become cool, perspiration breaks out over the forehead, and, instead of typhoid symptoms, the symptoms of collapse may ensue. If the disease is prolonged and the bowels are controlled, the symptoms of pyæmia may develop.

The anæmia that ensues is extreme, and there is great wasting. Convalescence is slow and may be attended by chronic diarrhoea. Before it is established, ulcers of the skin may form on various parts of the surface of the body. Arthritis is of common occurrence, and peripheral neuritis causing paralysis may occur during convalescence. Chronic dysentery may succeed the acute type. It is thus seen that the attacks may be of moderate severity or extremely grave; during the course of the latter, gangrene of the lower bowel may take place.

Diagnosis of Acute Dysentery. *H. D.* Exposure to cold and wet.

S. D. Rectal pain and distress.

O. D. No hepatic or vesical abscess.

P. D. Examine stools.

L. D. Serum diagnosis.

Tropical Dysentery. This form of dysentery may be due to *Bacillus dysenteriae* or to *Amœba coli*, and may be acute or chronic. The symptoms of the acute form are like those of acute catarrhal dysentery. In the chronic form the patient wastes, the complexion is earthy, the abdomen scaphoid, and the temperature afebrile. The intestinal symptoms are like those of chronic inflammation of the bowels. Secondary abscesses do not occur so frequently as in amœbic dysentery. The blood-serum agglutinates a pure culture of the organism. The diagnosis of the nature of the dysenteric process is based on the agglutinating power.

Diagnosis. *H. D.* Climate, exposure, epidemic.

S. D. Abdominal pain, no chills, rectal tenesmus.

O. D. Facies, anæmia, arthritis.

P. D. Negative.

L. D. Serum diagnosis.

D. D. Enteritis.

Amœbic Dysentery. This differs from catarrhal dysentery in many

respects. The onset may be abrupt or gradual, as in the previous form, with symptoms of intestinal catarrh. In most of the cases a frequent and painless diarrhœa follows a period of slight ill health. The diarrhœa alternates with short periods of constipation; the stools are watery and contain mucus, but no blood. The course of the disease is irregular. There may be intermissions and exacerbations of the diarrhœa without obvious cause. Amœbic dysentery may rapidly pass from one grade to another, or become chronic. One form is the gangrenous, which may scarcely be suspected from the symptoms until autopsy shows it to have been present. True relapses are common, and the tendency to chronicity is great.

The *milder* cases are attended by weakness, emaciation, and pallor; the expression is dull; the skin is dry and sallow; the tongue pale, flabby, moist, and slightly furred; the abdomen is normal or retracted; the temperature does not rise above 100° F., and the pulse-rate ranges from 70 to 90 per minute. Sleep is disturbed by frequent evacuations of the bowels.

In the *grave form* the face is drawn, or cyanosed, or flushed; the expression anxious; the mind is clear. Anorexia, intense thirst, and sleeplessness are present. The abdomen is greatly retracted, and there may be free sweating. The temperature is normal or subnormal, the pulse small and rapid. Progressive anæmia and loss of flesh are prominent and dominate the intestinal symptoms. The skin is dry and harsh, and of a dull greenish-yellow color if the cases are protracted.

The special features of amœbic dysentery are:

1. **The Anæmia.** This is due to diminution of the red cells and the hæmoglobin, first, because of the action of the amœbæ upon the red blood-corpuscles, which they destroy; second, the direct loss of blood; and, third, malnutrition. The first is the most prominent.

2. **Diarrhœa** may be the only feature of the disease. It is characterized by great variation in character and frequency in all grades and during different periods of the disease. Intermissions and exacerbations may be observed at any time. The latter begin suddenly, and subside in the same manner. They last from two to ten days. The intermissions continue from one day to three weeks, during which the feces are soft, but contain mucus. Councilman and Lafleur have observed this periodicity to be most marked in cases complicated with hepatic abscess.

3. **The Stools.** The reaction is generally alkaline. The stools are extremely variable according to the severity of the ulceration, and also vary in number and character from day to day in individual cases. In the gangrenous form they number thirty or forty in twenty-four hours at first, then decrease, so that toward the end of fatal cases but three or four take place. At first the movements are small, and consist of mucus with more or less bright blood and small fecal masses. As ulceration advances, the stools change, they become more copious and watery, feces are absent, and blood is not so frequent. Shreddy masses of grayish or yellow color, mixed with mucus, appear. If there is sloughing, they become greenish or grayish, resembling spinach, or reddish brown and very liquid or pultaceous. The odor is penetrating and offensive. Shreddy masses of necrotic

tissue are discharged. Gray liquid movements, somewhat slimy, contain more pus than the others. Small opaque, or translucent, gelatinous grayish masses, 1 to 3 c.mm. in diameter, are found in the stools.

In the more moderate types if the attack is abrupt, the stools at the outset are like those of gangrenous dysentery; if gradual, the stools are fecal, liquid, containing mucus and streaks of blood and many gelatinous grayish masses. Stools of this character number from four to ten in twenty-four hours; and may continue thus for weeks. During the exacerbations the stools resemble those of the second period of the gangrenous form. In chronic dysentery there is not so much mucus or blood, except in exacerbations. The stools are of the consistence of thin gruel and have an earthy or dull-yellow color. Mucus is persistently present, however, in the intermissions, when the stools are soft and fecal.

MICROSCOPICAL EXAMINATION. In the mucoid and bloody stools of the acute stage red blood-corpuscles, leucocytes, and large, round, or oval epithelioid cells are seen. The latter are sharply outlined and occur often in groups of three or more; the nucleus is about the size of a red blood-corpuscle, the protoplasm granular. They may be mistaken for amœbæ; but are non-motile and refract light less strongly. *Cercomonas intestinalis* is present, but bacteria are not abundant. In the later periods the cell-elements are less numerous; shreddy and muscular detritus and bacteria are observed, with elastic tissue-fibres. Charcot's crystals and phosphates are seen. In chronic dysentery the cell-elements are still fewer and amœbæ are easily detected.

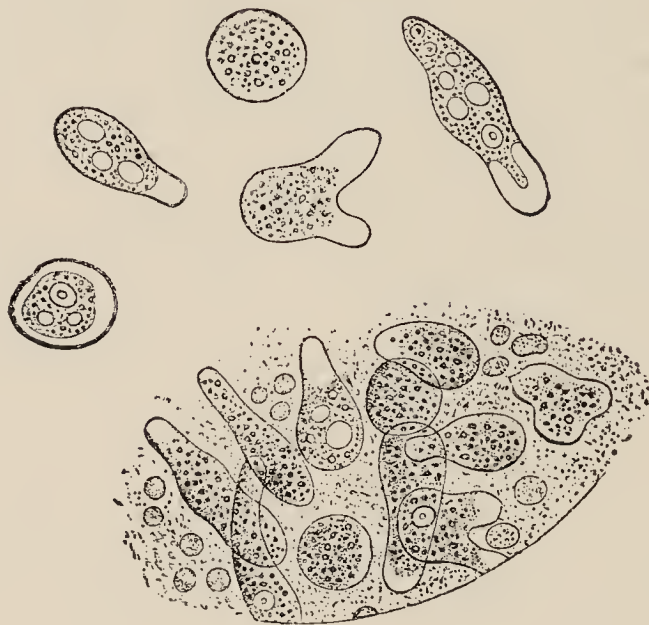
Amœba Dysentericæ. Amœbæ are found at all periods of the disease. They vary in different cases and at different periods in proportion to the severity of the intestinal ulceration. (See section on the Feces.) They are most abundant in the grayish-yellow gelatinous masses, next in the particles of clear or opaque mucus, and least in the fluid portions of the stools. In chronic dysentery they are found in all portions. In the intermission of the diarrhœa they may be found in the particles of mucus adherent to the feces. They disappear as recovery proceeds, although they may be seen after the evacuations have become normal. They vary in size and activity, and are most common in the alkaline and neutral stools; in acid stools they are scarce and rarely motile. In the more active forms of the disease red corpuscles are seen in the stools.

For the *detection of amœbæ* the following rules should be observed: First, the stools should be passed into a warm bed-pan and kept at a temperature of 30° to 35° C. until an examination is made. Better still, a soft warm catheter should be passed up the rectum. Mucus may then be withdrawn through the eye of the instrument. Second, the stools must be examined before they become acid. Third, the gelatinous masses in the stools should be selected for examination, for they contain the amœbæ in greatest abundance. A magnifying power of 400 diameters is required, although they may be seen with less. A $\frac{1}{12}$ oil-immersion lens is the best.

Histologic Appearance. When *inactive* the amœbæ are round or slightly oblong, highly refractive, and contain clear vacuoles which vary from small points to one-third of the diameter of the amœbæ. The ectosarc

and endosarc may or may not be sharply divided. If they are, the outer is hyaline or homogeneous, the inner is more refractive and contains vacuoles. They are difficult to recognize in this condition, being easily mistaken for swollen connective-tissue cells. The amœbæ frequently enclose red corpuscles, pus-cells, blood-pigment, bacilli, and micrococci.

FIG. 321.

*Amœbæ coli.* (HALLOPEAU.)

In a fresh state the nuclei can not be made out because they resemble vacuoles. The endosarc is not granular, is composed of a dense substance, and is highly refracting. When *active*, the movement is characteristic. It may be slow or rapid, and is of two kinds, a progressive movement and one limited to the throwing out of pseudopodia. The movements appear to be rhythmical in some cases, occurring at regular intervals. The movement is sudden and characterized by change in form of the pseudopodia. The ectosarc and endosarc are usually clearly defined. The pseudopodia are hyaline and homogeneous, like the ectosarc. The amœba changes its position sometimes by enlargement of the pseudopodia, into which the inner contents of the older part follow. The movements are increased when the examinations are made on the warm stage. These amœba may be stained with various aniline dyes.

In *catarrhal dysentery* the stools are uniform in character, quantity, and frequency. The onset is sudden, and evacuations consist of bright blood and viscid, clear mucus mixed with fecal matter. Soon they are composed entirely of viscid mucus and a little blood. In a week or ten days the mucus changes and becomes grayish white in color—is less blood-stained and brown; pultaceous or fluid fecal matter appears in the stools. As the blood and mucus disappear, formed feces return. In protracted cases there are soft, yellowish-brown or greenish stools in addition to the bloody mucoid stools. The frequency is greatest at the onset, and progressively diminishes until convalescence is established. The more frequent the evacuations the smaller the size of the stools. The mucoid stools are small, pultaceous, more bulky. On microscopical examination red and white corpuscles, cylindrical, epithelial, and oval epithelioid cells are seen. The latter are very characteristic, and occur singly or in groups.

Bacteria are more common as improvement sets in. In the pultaceous stools the cell-elements are scarce.

In *diphtheritic dysentery* the stools are watery. They resemble wheat-washings—evacuations such as are described in cases of *gangrenous dysentery*. They are grayish green or reddish brown and very offensive. Mucus is present in small amounts. At first they contain unclotted blood; later, minute dark-red clots. Shreddy and finely divided material, gray or reddish brown in color, is present, but there are no sloughs. The stools are not numerous at first, and average from seven to fifteen daily during the course of the illness; the quantity is small. Cylindrical epithelial cells are most abundant on microscopical examination. Red blood-corpuscles and leucocytes are observed, but fibrin constitutes the larger portion of the stool. In all the stools bacteria are present in great numbers.

Other Symptoms of Amœbic Dysentery. Abdominal *pain* is constant; it occurs in the early stages of both forms and in acute exacerbations. As the movements diminish, the pain decreases. In the gangrenous form pain also disappears, although the intensity of the process is increasing. In chronic cases the colic is complained of during the exacerbations; during the intervals there is a dull, aching or burning pain in the upper quadrants. In all cases the pain is cramp-like, boring or burning in character, and usually precedes and accompanies movements of the bowels. When severe it is general; but it is usually localized in the lower abdominal zone. Moderate tenderness on pressure is present in most cases along some part of the course of the large bowel. In catarrhal dysentery tenesmus is common; in the amœbic form it is infrequent. A burning sensation is generally present in the rectum and at the anus during and after the passage of feces. Nausea and vomiting occur at the outset or at irregular intervals, being caused by improper food, or due to complications. Hiccough occurs in the terminal stages.

FEVER In amœbic dysentery, fever is not a prominent feature, although there is usually a moderate rise in temperature. In the gangrenous form it is normal, or may be subnormal for days. Chronic dysentery is afebrile. In exacerbations of diarrhoea slight fever may occur. Complications cause a higher temperature. If fever is present, it may be remittent or intermittent in character; or if the illness is prolonged, first continuous, then remittent, and then intermittent. If the latter, the usual morning fall is observed, although an inverse temperature may be present. Rigors occur with the complications. In the gangrenous form, sweating is observed, with subnormal temperature. In cases of abscess the fever is intermittent or remittent.

In *chronic dysentery* the skin is excessively dry. The circulation and respiration are influenced by the pyrexia. Anæmia is pronounced. When exhaustion ensues, the pulse becomes more feeble, compressible, and rapid. The urine is albuminous, and often contains casts. In the gangrenous form there may be retention of urine.

Complications. The complications of amœbic dysentery are: 1. Hepatic abscess or hepatopulmonary abscess. 2. Peritonitis. 3. Hemorrhage from the bowels.

HEPATIC ABSCESS. This complication may develop at any period of the disease. The time of the disease when it occurs can not be determined definitely. In the subacute cases it is liable to develop from the fourth to the twelfth week. The abscess may develop on the convex surface of the right lobe of the liver near the coronary ligament. The lung also becomes involved. Councilman and Lafleur suggest that infection takes place by the peritoneum. (See Abscess of the Liver.) The symptoms of abscess of the liver will be treated under the section devoted to liver disease, but it is important to note that hepatic symptoms may occur in cases in which, on account of the mildness of the disease, the local bowel trouble may be overlooked.¹ If the association of hepatic pain with fever and discharge of mucus from the bowels is observed, it is barely possible, even if an examination of the feces can not be made, that an hepatic abscess is present. If, in addition, cough and expectoration occur, involvement of the lungs is possible.

HEPATOPULMONARY ABSCESS. The character of the expectoration points conclusively to the nature of the lung complication. After a period of dry, hacking cough, sudden expectoration of mucopurulent or bloody sputum takes place. It is of a dirty-red or brownish color, not unlike anchovy sauce. From this time on, this material is expectorated in varying quantities after a paroxysm of coughing. The expectoration is diffuent, tenacious, and frothy. It varies in color from bright red to russet-brown; it may be bile-stained; the reaction is alkaline; the odor is not putrid. At a later period it becomes more purulent, and contains less blood. The sputum separates into three layers: an upper frothy layer, a middle layer of turbid fluid, a thin layer of mucopus below. Large amounts may be coughed up in twenty-four hours. The sputa contain on examination blood-corpuscles, leucocytes, round alveolar epithelial cells, and polyhedral, fatty, degenerated cells which look like liver-cells. Elastic tissue fibres from the lungs are found with crystals of hæmatoidin and tyrosin, and Charcot's crystals. Bacteria are present. Amœbæ are constantly present. They vary in size and activity, but are larger than those seen in the stools. The sputum should be kept warm and examined as soon as possible. Abscesses form in other situations, as the jaw. (Flexner.)

PERITONITIS. Peritonitis from perforation is not a common complication of amœbic dysentery, but takes place occasionally in the gangrenous form. Peritonitis without perforation may occur. The symptoms do not differ from peritonitis under other circumstances.

Hemorrhage from the bowel occurs and may be sufficiently profuse to cause death. This accident may occur in the course of amœbic abscess of the liver, as in a case reported by the author, in which there were no intestinal symptoms. Other complications which have been described under catarrhal and croupous dysentery are likely to occur in this affection.

Diagnosis. *H. D.* Hygienic surroundings; climate; temperature; food; country; clinical course.

S. D. Enteritis; toxæmia.

¹ See "Amœbic Abscess of Liver." Musser and Willard, Trans. Phila. Co. Med. Soc.

O. D. Negative.

P. D. Liver abscess ; pubic fistula ; amœba.

L. D. Feces ; sputum ; pus from abscess.

D. D. Malaria.

The diagnosis of amœbic dysentery is rendered absolute by finding the amœbæ in the stools. The history and the course of the illness must also be taken into consideration, the characteristics of which have been previously detailed. The irregularity and the intermittency of the diarrhœa, the infrequency of tenesmus, the moderate fever, the reaction of the stools and their comparative freedom from bacteria are further corroborative points.

The Plague.

Plague is an acute, specific, infectious and contagious disease, occurring in epidemics, characterized by high fever, sometimes by petechiæ and other hemorrhages, and, in cases which last long enough, by buboes. The death-rate is extremely high. It is a disease of the East, being endemic in some parts of India ; but epidemics have occurred in Italy, Russia, China, Turkey, England, and other parts of Europe.

Incubation. The period of incubation is from two to seven days. The invasion is marked by lassitude, languor, headache, and dizziness. The stupid aspect and staggering gait may lead to the belief that the patient is drunk. Chill or chilliness soon supervenes, followed by fever, which often rises to hyperpyrexia, and is accompanied by unquenchable thirst, and sometimes nausea and vomiting. Delirium and a typhoid condition follow, with a marked tendency to failure of the circulation and collapse. If the patient survive until the second or third day, glandular swellings develop in the groin or axilla, or at the angle of the jaw. Often they have to be sought for to be found. Sometimes they are prominent, and are followed by suppuration and even ulceration. Carbuncles are much rarer manifestations than buboes. Petechiæ, vibices, hemorrhages into the kidney, and bloody vomit occur in the worst cases.

The clinical divisions are those of *pestis minor*, in which fever, some glandular swelling and possibly suppuration occur ; and *pestis major*, in the course of which buboes, septicæmia, the typhoid state, pneumonia, and other local infections occur. The cases are often ambulant, but the urine and feces contain bacilli.

Diagnosis. *H. D.* Exposure ; climate ; epidemic.

S. D. Clinical course. Symptoms of toxæmia.

O. D. Buboes ; carbuncles ; hemorrhages.

P. D. Pneumonia if present.

L. D. Organism in pus ; sputum ; blood. Serum diagnosis.

D. D. Typhoid fever ; other infections.

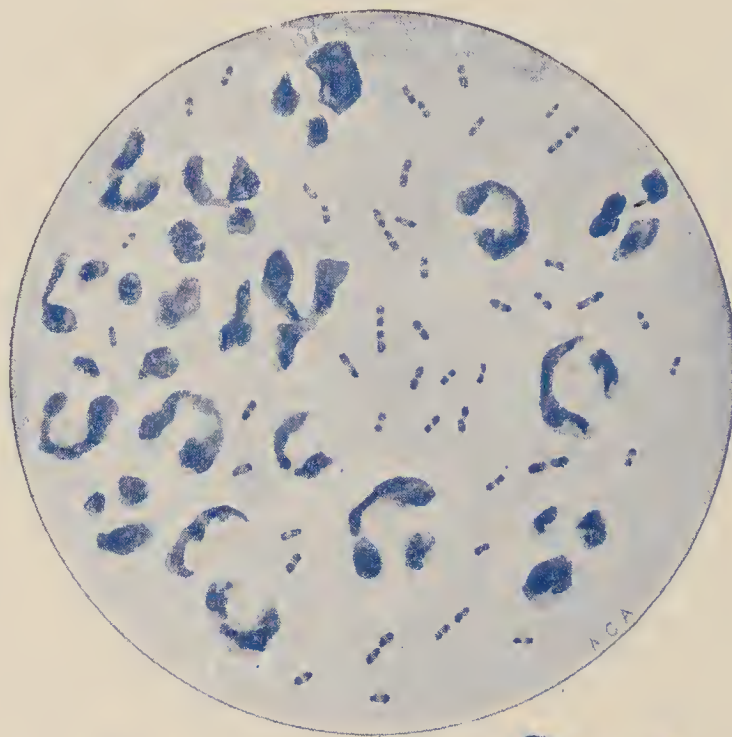
The diagnosis is based upon the history, the clinical course, and the results of bacteriological examination. The following description from Abbott enables the diagnosis to be readily made :

“This organism is described as a short, oval bacillus, usually seen single, sometimes joined end to end in pairs or threes, less commonly as

longer threads. It stains more readily at its ends than at its centre. It is sometimes capsulated; is non-spore-forming; is aërobic, and is non-motile. It is found in large numbers in the suppurating glands, in the sputum, and in much smaller numbers in the blood. (See Fig. 322.)

FIG. 322.

A



B

Bacillus of bubonic plague: *A*, in pus from suppurating bubo; *B*, the bacilli very much enlarged, to show peculiar polar staining. (ABBOTT.)

“It is demonstrable in cover-slip preparations made from the pus and in sections of the glands by the ordinary staining methods. Yersin states that it retains its color when treated by the method of Gram, while Kitasato says that it at one time stains by this method and at another it becomes decolorized. Aoyama observed that those bacilli within the suppurating glands were decolorized, while those in the blood retained the stain when treated by Gram’s method.”

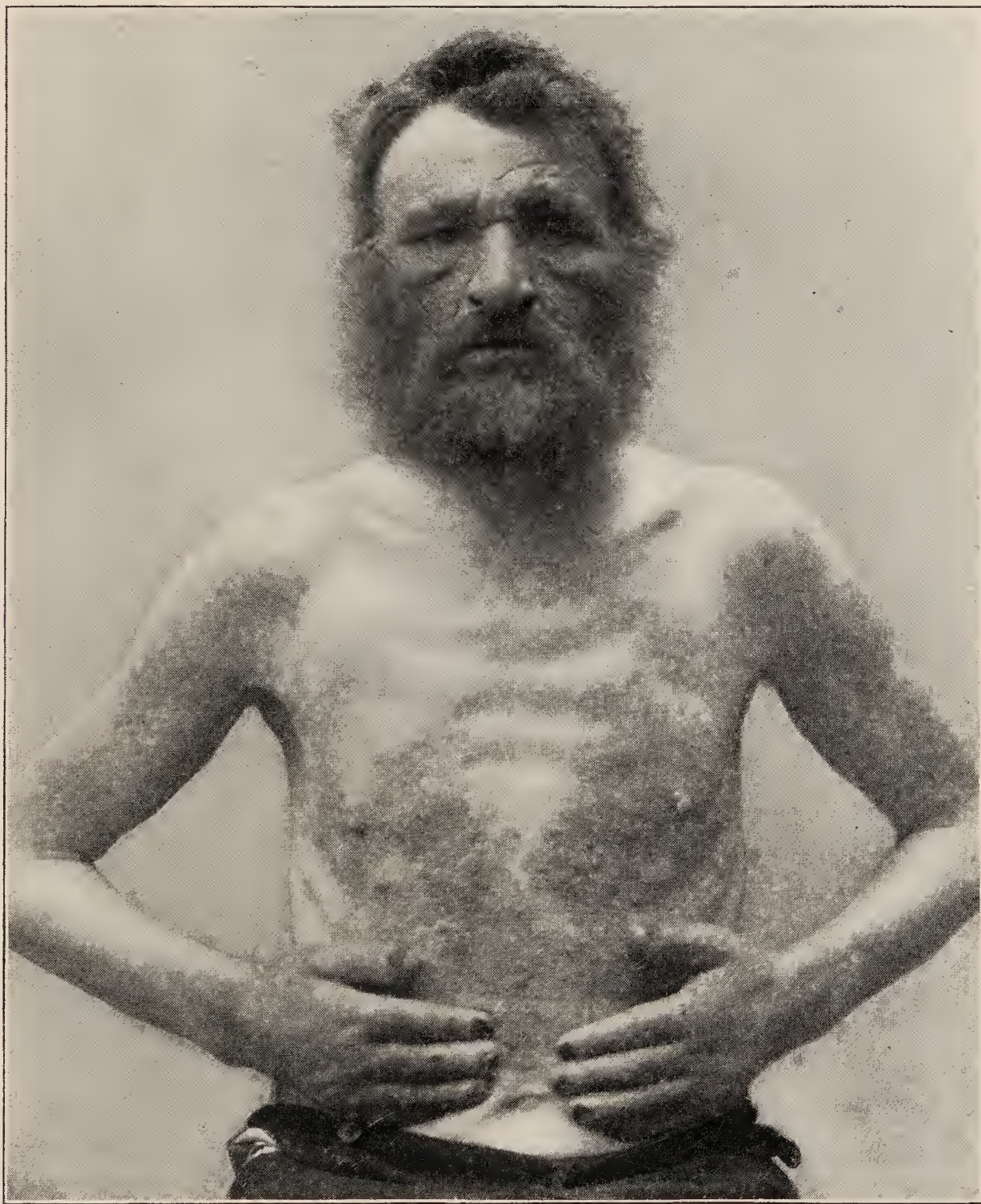
The duration is from six to ten days. If there is much suppuration, convalescence is prolonged.

Leprosy.

A chronic, specific, infectious disease, characterized by the development of tubercles, anæsthetic patches, and neuritis, and followed by ulceration and destruction of tissue. The disease occurs especially from puberty to the thirtieth year, and oftener in men than in women. It develops slowly and insidiously. Sometimes the first skin-lesion is a crop of bullæ, suggestive of pemphigus. More commonly there appear reddish or violet-colored patches, varying in size from $\frac{1}{4}$ inch to 2 or 3 inches in diameter, and becoming of a darker hue later. The next step

is the formation of nodules, which are characteristic of the disease. These may develop upon the patches already described, or in other places. They vary in size from that of a pea to that of a bird's egg or larger. They are most common upon the face and extensor surfaces of the arms, legs, fingers, and toes. The tubercles consist of an infiltration into the true skin; they are raised, firm, relatively painless, and vary in color from red to copper. The face is characteristically distorted into a fierce expression (leontiasis). The tubercles may become absorbed and leave

FIG. 323.



Leprosy.

atrophic areas, but generally they break down into eroding ulcers, which slowly burrow and increase in extent, eating off a portion of the nose, fingers, hands, and feet, and exposing muscles, tendons, nerves, blood-vessels, and bone. Tubercles form also upon nerve-trunks, and ulcers upon the mucous membranes. (See the Nose and Larynx.)

In other cases, or in combination with the tubercles, especially upon the limbs and trunk, there are *anæsthetic* areas. Ulcers may follow with-

out the previous occurrence of tubercles. With the anæsthetic patches are associated crops of *bullæ* and *neuritis*.

The further peculiarities of the disease are : its long duration, its slow progress interrupted by apparent healing of some of the ulcers ; its afebrile course (the temperature is generally subnormal) ; its comparative painlessness, and the slight impairment of the general health.

Death results from gradual wasting, or is hastened by some intercurrent affection.

Diagnosis. *H. D.* Exposure ; residence.

S. D. Painless lesions.

O. D. Skin-lesions, nodules ; ulceration ; anæsthesia ; facial expression ; afebrile.

P. D. Negative.

L. D. Bacilli in lesions.

D. D. Syphilis ; hysteria.

The specific cause of the disease is *Bacillus lepræ* of Hansen. It is found in the thin pus of the ulcers and in the lesions. It consists of rods 4μ to 6μ long and 1μ broad, closely resembling the tubercle bacillus, and stains in alkaline fluids, but does not bleach after exposure to acids. Staining cover-slip preparations with the Ziehl-Neelsen fluid and decolorizing in acid and alcohol bring the bacilli out quite distinctly. They may be distinguished by the fact that they yield their color more readily, and easily take aniline dyes in simple watery solution. (Von Jaksch.)

The *diagnosis* from a tubercular *syphilide* is made by the history of the case, the possibility of infection, the bacteriological examination, the slow progress, and the inadequacy of specific treatment. Anæsthesia and neuritis point to leprosy, although their occurrence in young neurotic subjects also suggests hysteria.

Actinomycosis.

The general symptoms attending this infection are like those of suppurative infections. The *fever* is irregular, often intermittent. It is a specific, infectious disease of cattle, occurring occasionally in man, attacking especially the lower jaw, lungs, and intestines, and characterized by a long duration, by the development of tumors and metastatic growths, and by *pyæmic* symptoms.

Actinomycosis is due to actinomyces, or ray-fungus (see Fig. 325), which produces in cattle the disease known as big or lumpy jaw and swelled head. The fungus is conveyed in the food or drink, and gains entrance to the body through abrasions in the mouth or a decayed tooth, or is inspired into the lungs. Israel, Ponfick, and Boström have given us the greatest amount of information in regard to this parasite. It was discovered in 1845, in human beings, by B. von Langenbeck, and in 1877, in cattle, by Bollinger.

At the seat of invasion a slowly growing, slightly painful tumor develops. Bones are affected as well as soft tissues. These become swollen and suppurate, the fungus being at all times obtainable. The fungous

masses appear to the unaided eye as particles of yellow sand, and are greasy to the touch.

Pulmonic Form. Actinomycosis of the lung may be divided into three stages; a latent stage, when the lung proper is affected; an active stage, when extension to the pleura and chest-wall takes place; and a final or chronic stage, when perforation and the formation of a thoracic fistula occur and the adjoining organs become affected.

FIG. 324.



Case of actinomycosis.

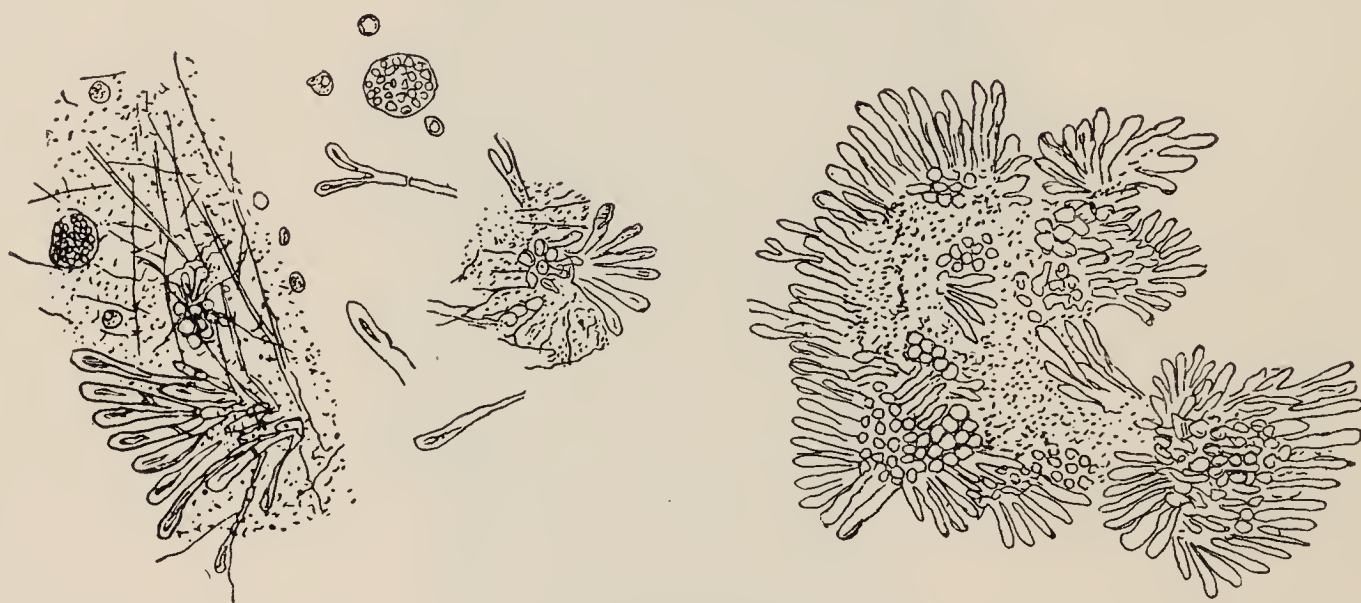
The symptoms of the *first stage* are those of chronic bronchial catarrh, with later the occurrence of the physical signs of consolidation, especially in the mammillary and axillary regions of the chest, in the middle zone of the thorax. The apices and bases are rarely affected primarily.

The symptoms of the *second stage* are those of pleurisy, with adhesions and with or without effusion. At this time the disease may extend downward to the liver and peritoneum, or the pericardium may become infected. Fever and pain accompany these processes. On physical examination, in addition to the signs of the pulmonary and pleural conditions above mentioned, swelling of the thoracic wall will be observed, not unlike that due to an empyema which is about to perforate. The swelling, which is at first dense, and hard, and red, becomes softer in small areas, and may fluctuate. Fluid, which is mucopurulent and contains the parasite, may be removed by aspiration. Repeated taps may fail before the needle finds the serous or sanguinoserous exudation in the pleura. The sputum at this time may accidentally contain the parasite, although this is rare; it is mucopurulent, but it is said never to contain elastic fibres. The course of the disease at this time may extend over many months, in contradistinction to empyema on the one hand and carcinoma on the other.

In the *final stage* ulceration of the swelling is seen in many places, a fistula forms, and the disease extends to adjacent structures.

Metastasis. Secondary infection may occur and symptoms of pyæmia develop. The masses which form upon the intestinal mucous membrane may lead to suppuration and perforation of the intestine. Metastasis to any organ may occur, with resulting local symptoms. The duration depends upon the organs involved in metastases. If metastases do not lead to early death, that result is brought about at the end of months or years by slow pyæmia, with resulting amyloid degeneration and its consequences.

FIG. 325.

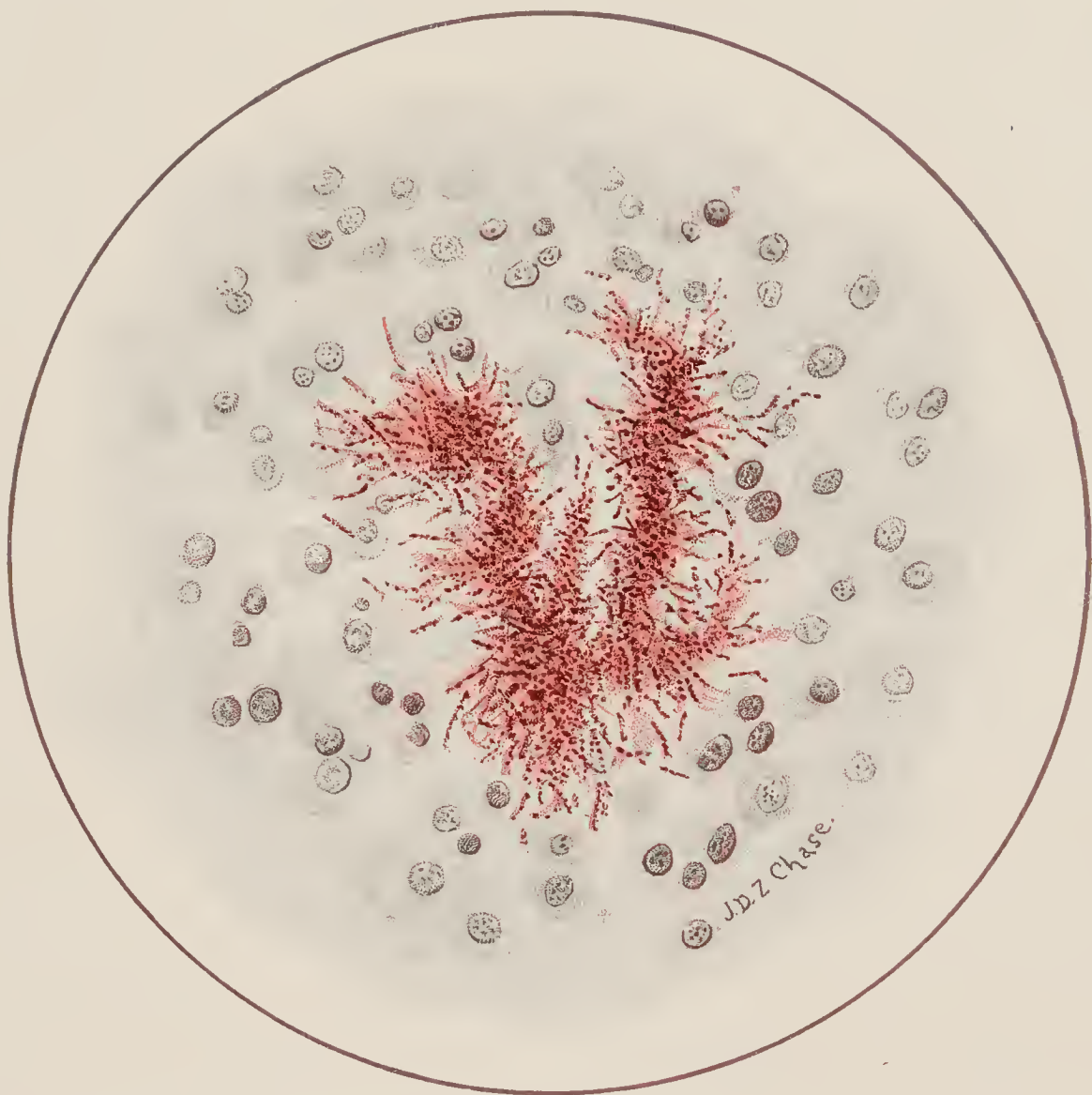


Actinomyces.

Character of the Pus. Actinomycosis is usually associated with chronic inflammation and the production of pus. The pus is peculiar. It is thin and viscid. Nodules of gray or yellow color, the size of a poppy-seed, can be seen in it with the naked eye when it is spread on a slide. With a low power these particles appear as aggregations of spherules, which with a higher power are seen to be arranged in masses radiating from a common centre. Each separate spherule is pear-shaped. They have high refractive power. The centre of the masses is occupied by a network of fibres. If the mass be broken up, numerous club-shaped forms are seen at the periphery, while at the centre a sort of detritus alone is observed. The micro-organism belongs to the class of fission-fungi, and the club-shaped bodies are the degenerated forms. (See Fig. 325.)

Gram's method of staining brings out the threads of the network most distinctly. The centre is made up of a network of minute spherical organisms, with converging, constituent threads. The whole is surrounded by a delicate envelope. The pear-shaped bodies may be defined by Weigert's process. Make a solution of 20 c.c. of absolute alcohol, 5 c.c. of concentrated acetic acid, 40 c.c. of distilled water, and sufficient French extract of litmus to color it ruby-red after repeated filtering. In this solution the cover-glass preparations are allowed to remain for an hour, and then rinsed with alcohol rapidly and placed in a 2 per cent. gentian-violet solution for three minutes. The fluid should be boiled

PLATE XVIII-a.



Section through wall of brain abscess showing character of streptothrix colony. Stained with hæmatoxylin, followed by modified Gram-Weigert method, 1/12 in. homog. immersion objective.

before use and filtered after cooling. The fungous threads are stained a ruby-red, while the central mass of actinomyces is colorless.

Diagnosis. *H. D.* Diseased teeth or mouth, exposure.

S. D. Painful indolent swelling; symptoms of empyema or bronchitis.

O. D. Swelling about jaw slowly suppurates; bone disease; metastases; pyæmia; enteritis.

P. D. Some cases; empyema; bronchopneumonia.

L. D. Pus; smears; sputum; exploratory puncture; ray fungus.

D. D. Suppurations; pulmonic affections; enteritis.

Simple *microscopical examination* is usually sufficient to determine the nature of the fungus. The recognition is more positive if we bear in mind the peculiar character of the pus in which the nodules and the club-shaped forms are seen. It must not be mistaken for the radiating leptothrix threads found in the mouth. Pure cultures have been obtained resembling macroscopically those of tubercle bacillus.

Streptothrical Infections.

The pathogenicity of streptothrices and their relation to infections and lesions described are very evident, though cultural and animal experiments are lacking in some of the earlier observations. It is clear that actinomycosis and Madura foot can no longer occupy their isolated positions as examples of streptothrical infections. The predilection of these organisms for certain regions or systems of the body is very apparent, the *lungs*, the *brain*, and the *skin* being most frequently affected; the brain, however, is more often invaded by metastasis in cases of primary pulmonary or bronchial gland infections.

The *pulmonary lesions* in which the streptothrices have been found either alone or with other organisms show considerable variety: bronchopneumonia, extensive consolidation, abscess, bronchiectasis, empyema, and necrotic bronchitis.

The lesions in the *nervous system*, practically always metastatic infections from pulmonary or bronchial gland disease, have been abscess, meningitis, or large areas of softening from thrombosis.

An *abscess* of the *kidney* was noted in one instance. Naunyn found in his case an *endocardial excrescence* containing a streptothrix.

From a clinical standpoint but little that is distinctly new is presented in streptothrical infections. The cutaneous lesions of Madura foot, the cases of Rosenbach, Ferri and Faguet, Scheele and Petruschky, the cutaneous expression of actinomycosis, the actinomycotic-like skin infection of Fullerton, show distinct differences. The pulmonary infections with their secondary accidents are all more or less hidden under the mask of a tuberculous process, herewith termed *pseudotuberculosis*, or resemble some one or other of the number of pulmonary lesions, such as *abscess*, *gangrene*, or *bronchiectasis*, but the detection of the streptothrix and its mycelial masses is as diagnostic as the demonstrations of tubercle bacilli. Not all instances reported have been primary infections. In many the streptothrix may have been a very late invader. Many instances of streptothrical infections have probably passed unrecognized,

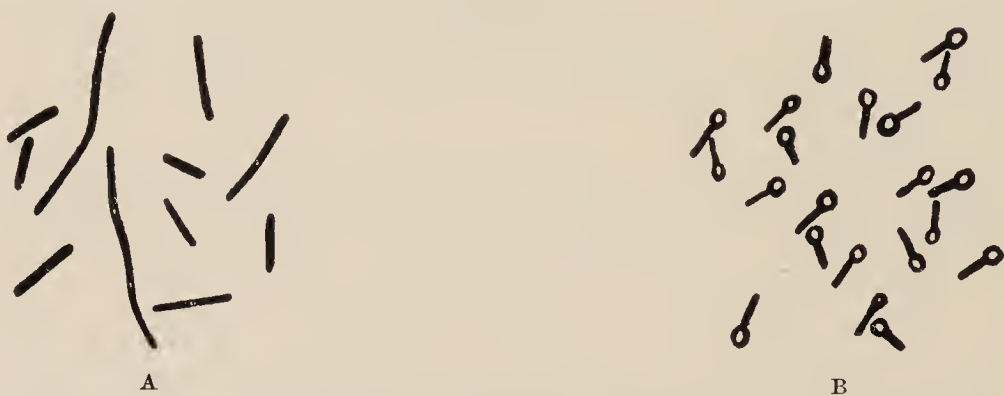
owing to the peculiar tinctorial and cultural reactions of the micro-organism. Rullman, Ferri and Faguet, Aoyama, Scheele and Petruschky were able to determine the nature of the disease in their cases by the findings in the sputum, as was Gwyn in the case I have reported.

Tetanus.

Tetanus is an acute infectious disease of the nervous system, the essential characteristic of which is persistent tonic spasm of the muscles of the jaws (*lockjaw*) and of the spinal and thoracic muscles.

The disease begins with stiffness of the jaws, which steadily increases until within a few hours there is complete tonic spasm. The neck muscles, and then those of the spine and trunk, become rigid, so that the body is arched backward and may rest upon the heels and head (*opisthotonos*). The facial muscles share in the spasm, and by their contraction produce a horrid, grinning countenance (*risus sardonicus*). The contracted muscles become painful, and there is also epigastric pain. The rigidity is persistent, but is interrupted by exacerbations in which the phenomena already described are exaggerated, and, in addition, respiration is embarrassed, the face becomes livid, the skin bathed in sweat, and the patient is further distressed by increased pain in the affected muscles. The body may be bent forward (*emprosthotonos*) or laterally (*pleurosthotonos*). The temperature is not constant. It may remain normal, be moderately elevated, or hyperpyrexia may be present, especially toward and after the end in fatal cases. The spasm ceases during sleep, but subsequently returns.

FIG. 326.



Tetanus bacillus: A, vegetative stage, from gelatin culture; B, spore-stage; showing pin-shape. (ABBOTT.)

Bacteriological Diagnosis. The cause of the disease is the bacillus of tetanus, which produces the convulsive poison *tetanin*. The bacillus is seen as a delicate, slender rod, with a terminal spore. It stains with aniline dyes and Gram's fluid. Cultures may be made with the pus.

Tetanus frequently follows an injury. *Trismus neonatorum* and *puerperal tetanus* are names given to special varieties which occur in new-born children and in puerperal women. Tetanus is much more common in men than in women, and Gowers states that three-fourths of the cases occur between the ages of ten and forty. It is much more common in hot than in cold countries, though cold is an exciting cause.

In traumatic and puerperal cases the disease usually develops in from

a few days to two weeks from the time of injury or childbirth or abortion. In new-born children it occurs usually during the first week. It lasts from two to six weeks, but may be fatal much earlier, or in rare cases last even longer.

Diagnosis. *H. D.* Trauma ; infants ; puerperium ; climate ; season.

S. D. Pain in muscles.

O. D. Rigidity ; spasm (lockjaw).

P. D. Negative.

L. D. Smears ; cultures ; inoculation.

D. D. Strychnine-poisoning.

Tetanus must be distinguished from strychnine-poisoning. In the latter the jaw muscles are never involved early, if at all, and the muscles are relaxed between the paroxysms. It is distinguished from tetany by the history and the distribution of the spasm, which in tetany is confined to the extremities.

Trichinosis.

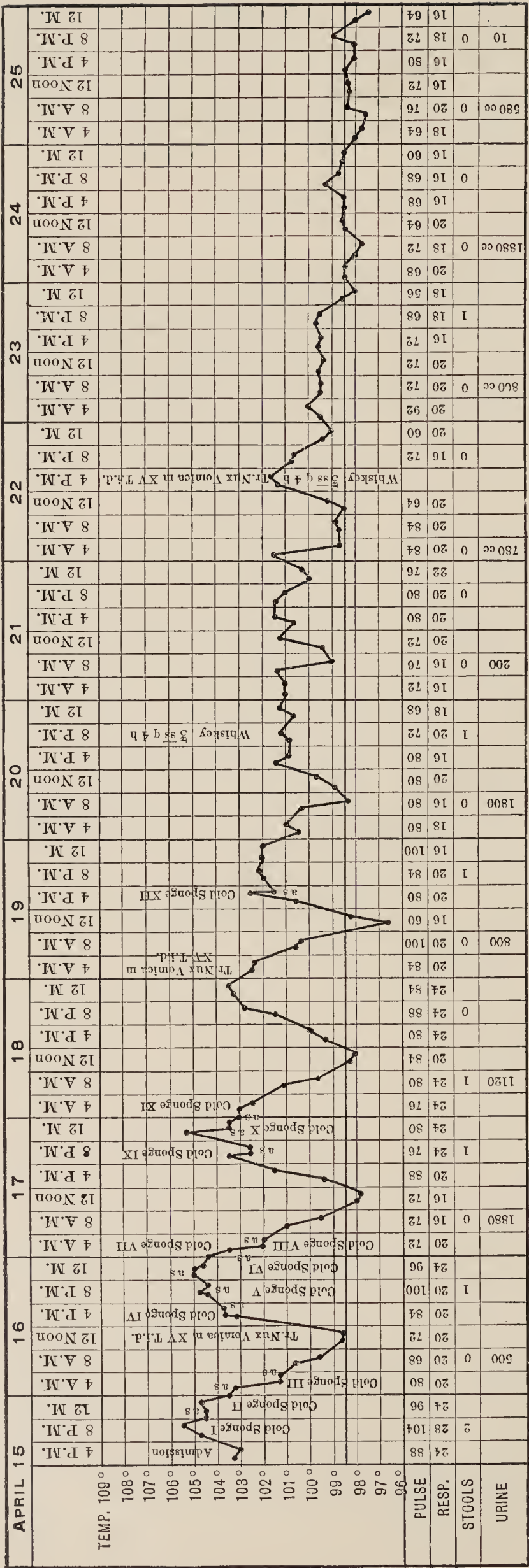
Definition. The infection is acute, caused by absorption of *Trichinæ spiralis*, and characterized by fever, gastric and intestinal irritation, followed by pain and stiffness in voluntary muscles, œdema of the eyelids, face, and feet, by profuse sweating, and by death or tardy convalescence.

The trichinæ are absorbed by human beings through raw or imperfectly cooked food, often in the form of sausage. The trichinæ are encysted when absorbed, but within forty-eight hours they are liberated in the intestine and can be found adherent to the mucous membrane. In the course of six or seven days each liberated female worm produces about 180 embryos, which immediately penetrate the walls of the intestine and travel or are carried to all parts of the body, becoming encysted in turn.

Clinical Course. Swallowing of trichinous flesh does not necessarily produce symptoms ; the trichinæ may be destroyed in the stomach, or, if calcified, may pass through the intestine unchanged. When symptoms result, the severity depends upon the number of trichinæ that become liberated. The symptoms are sleeplessness, lassitude, anorexia, nausea, vomiting, tenderness over the abdomen, and diarrhœa. Headache is a constant and marked symptom of invasion. Colicky pains attend the gastro-intestinal symptoms. These symptoms may not be marked in the beginning of the disease ; or they may be so severe as to cause death in two or three days. If the patient survive, toward the end of the week the voluntary muscles become stiff, painful, and contracted. The muscles feel hard and swollen. The eyelids, face, and sometimes the feet become œdematous. Depending upon the muscles involved, there are interferences with the eye movements, contractions of the jaw muscles, difficulty in breathing or in swallowing, etc. The calves of the legs are especially involved. Recurrent œdema over the affected muscles, eyelids, and face is very common and characteristic. Marked erythema of the limbs occurred in one case. Profuse sweating also is very common, and at times there are severe neuralgic pains. The later stages in fatal cases are marked by insomnia, delirium, stupor, and coma.

The *duration* varies from a few days to four or five weeks, or even

Fig. 327.



Intermittent fever in trichinosis. (OSLER.)

longer. Muscular pains may persist for months after recovery. Death results from exhaustion, or from some complication, as pneumonia or ulceration of the large intestine.

Fever. The fever is usually moderate, but it may be high. It is accompanied by malaise, with pains in the joints and muscles, preceding the true local muscle pain.

In some cases the temperature may be only moderately elevated for a few days, and then fall to normal, and even below normal, especially during convalescence. In other instances the temperature-curve may be markedly intermittent. The chart from Osler's monograph shows this peculiarity. (See Fig. 327.) Finally, the fever range is not unlike that of typhoid fever in many instances. Strümpell observes that the fever is seldom continuous for any length of time, and that its course is interrupted by frequent and prolonged intermissions. Niemeyer compares the curve to that of typhus, and Eichhorst to that of typhoid fever.

Pulse. The pulse is very frequent if trichinæ reach the heart.

The Blood. Brown, in studying Osler's cases, found an increase in the leucocytes, and on a differential count a great increase of the eosinophiles. The diagnosis of five of the six cases studied by Brown was suggested by the eosinophiles. The leucocytes were increased to 17,000 per c.mm. The eosinophiles increased from 2 per cent., the normal, to 37 per cent., and at one time to 68.2 per cent. In subsequent cases their average increase was as high as 48 per cent. Blumer, in the report of an epidemic, confirms the observation. Subsequent observations have confirmed these studies, so that *eosinophilia* is a recognized accompaniment of this parasitic invasion.

Diagnosis. *H. D.* Exposure to cause (food).

S. D. Pain in muscles.

O. D. Œdema, subcutaneous and muscular tubercles, rigidity, fever, sweating, gastro-enteritis.

L. D. Examination of muscle; leucocytosis, eosinophilia.

D. D. Differential from typhoid fever and muscular rheumatism.

The diagnosis is based upon the history, the peculiar *muscular pains* and *swellings*, the localization of the œdema, and the *leucocytosis* and *eosinophilia*. The muscles are swollen and hard, painful on pressure, and contracted. There is no involvement of the joints, an important point in the diagnosis. The œdema (see Chapter XXIV., Part I.) is seen in the eyelids and over the eyebrows. It is of common occurrence over the swollen and tender muscles.

Trichinosis is distinguished from *typhoid fever* by the presence of vomiting, and œdema of the face and eyelids, the development of muscular troubles, and by the absence of hebetude, delirium, and other typhoid symptoms, and absence of the characteristic eruption and of enlargement of the spleen. The Widal test, of course, is necessary.

Muscular rheumatism is distinguished by being limited to one part, as the lumbar region, arm, or chest; by its appearance following exposure to draught; and by the fact that it is not preceded by nausea, vomiting, and diarrhœa, nor accompanied by œdema.

CHAPTER II.

THE INTOXICATIONS.

FEBRILE INTOXICATIONS.

Sun-stroke.

SUN-STROKE (siriasis, thermic fever, insolation, heat-stroke), whether the cause be the direct action of heat upon the brain-centres, or the production of some toxic substance, is the most pronounced expression of *fever* apart from the infectious disorders. The flushed face, the pungent skin, the dyspnœa, and the rapid pulse forebode the high body temperature, which in the axilla may reach 108° to 112° F. in a very short time, and death takes place in coma from hyperpyrexia. If recovery takes place, the temperature may be moderate and continued for a few days. Nervous and cardiorespiratory phenomena are added to the picture. In some instances dyspnœa, heart-failure, and coma may follow rapidly, and death ensue in one or two hours. In other cases pain in the head, dizziness, and languor precede the stupor. Nausea and vomiting, perhaps diarrhœa, chest oppression, frequent micturition, and convulsions may precede insensibility. Unconsciousness is lost quickly or gradually, and it may be transient or pass into deep coma. Relaxation of the muscles with twitching is seen, and the pupils, at first dilated, become contracted. As the coma deepens, the heart's action becomes more rapid and feeble, the respirations hurried, shallow, and irregular, and death ensues, preceded or not by convulsions.

The diagnosis is based on the history, the mode of onset, and the hyperpyrexia. Thermic fever must be distinguished from uræmia and apoplexy.

Heat-exhaustion is readily recognized. The moist, pale, and cool skin, the soft, feeble pulse, the quiet but hurried breathing, are unattended by fever. The collapse, for such it is, is not attended by coma, and it usually responds to treatment.

Morphinism.

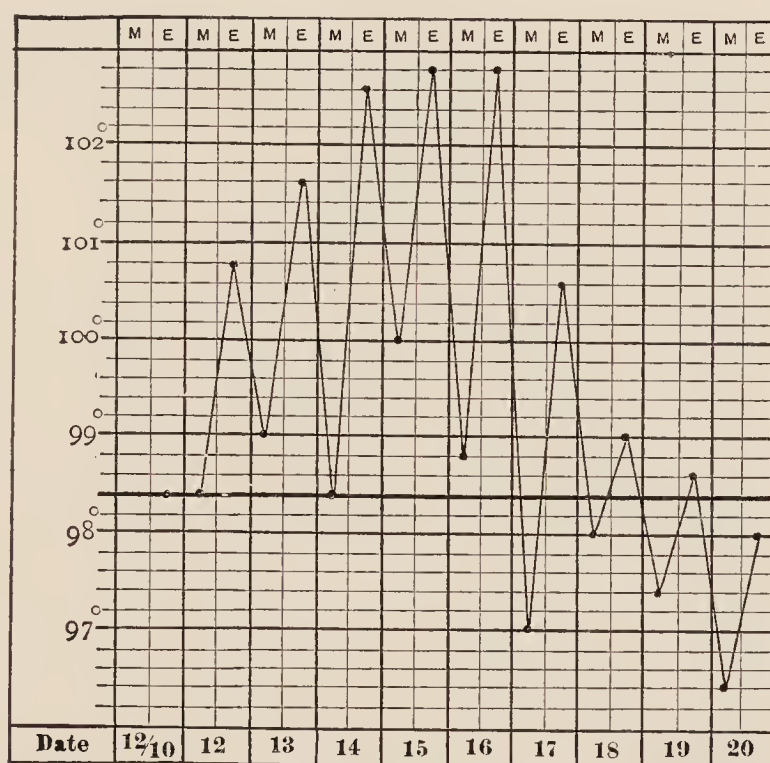
Lewin showed that morphinism is attended by fever. The fever may be continued or intermittent. When the latter, *chills* are of frequent occurrence. The diagnosis is based on the history, on the evidence of malnutrition without cause, on the general depression and lassitude, and upon the temperament of the patient, to which are added insomnia, restlessness, and itching of the skin. The peculiar sallowness of the complexion and the prematurely aged appearance are familiar. Pseu-

doneuralgic pains are common, tabetic symptoms may be present, and notably gastro-intestinal symptoms, as gastralgia, vomiting, diarrhœa (especially if the drug is withheld). Fever, it must be remembered, may be absent.

Simple Continued Fever.

A non-contagious fever, lasting from one to twelve days, not dependent upon any known specific cause, and not attended by definite local lesions. The chief feature is the continued elevation of temperature. It occurs especially in children and in those prone to ready disturbance of the heat-regulating apparatus. Great mental and physical exhaustion, prolonged bathing in the hot sun, and disturbances of digestion may cause it. Perhaps, as suggested by Guitéras, some of these cases occurring in the tropics and in very hot weather should be regarded as mild forms of thermic fever.

FIG. 328.



Simple continued fever.

The onset of the disease is abrupt. There may be a chill, or in nervous children a convulsion; but these are rare. The temperature rises rapidly from 102° to 104° F., accompanied by headache, thirst, restlessness or drowsiness, loss of appetite, a coated tongue, constipation, and occasionally nausea. The urine is scanty, and sometimes there is a heavy deposit of urates. There may also be more or less muscular soreness. Sometimes within twenty-four or forty-eight hours free perspiration takes place, with rapid subsidence of the fever. This is *ephemeral fever*.

In other cases the fever continues for a week or ten days longer. During this time the symptoms already noted continue. Sleep is disturbed and mild delirium is at times present. Respiration and pulse are not much accelerated. Sudamina upon the abdomen and herpes on the lips are common. Pale-bluish maculæ are sometimes seen. The spleen is not enlarged except in very rare cases, and there are no local evi-

dences of disease. The fever subsides more gradually than in ephemeral fever, the defervescence being marked at times by perspiration, a few loose stools, a copious deposit of urates in the urine, or by hemorrhages from the nose, rectum, uterus, or urethra.

The *diagnosis* from other fevers and febrile affections is made by the absence of any characteristic eruption, of enlargement of the spleen and liver, and of any lesion, such as endocarditis, bronchitis, or pneumonia.

FOOD INTOXICATIONS.

Among the intoxications which give rise to fever are those due to food-poisoning. Meat, milk-products, and shell-fish cause an intoxication of the system which often threatens life, and, from the suddenness of the attack and the severity of the symptoms, points to an infection rather than an intoxication.

The history of the case is often the first clue to its nature. The symptoms are those of acute gastro-intestinal irritation, to which are added, with or without afebrile periods, the symptoms of collapse.

Meat-poisoning.

In the intoxication arising from poisoning by meat, the temperature rises from 101° to 104° F., preceded usually by a brief period of chilliness. The occurrence of fever may be preceded by a period of incubation lasting from twelve to forty-eight hours. During the period of *incubation* there are malaise, loss of appetite, nausea, and colicky pains. As they increase chilliness ensues, and in some instances there is a marked rigor. Prostration occurs almost immediately, with giddiness and faintness, and the occurrence of cold perspiration. Headache and backache are liable to occur. Following the chilliness the symptoms of gastro-intestinal irritation arise, diarrhoea being more frequent than vomiting. The abdominal pain increases and the perspiration and clammy sweats become more pronounced. As further evidence of the intoxication there is an extreme degree of muscular weakness. The pulse becomes rapid, and later thready. In addition to muscular weakness, cramps in the legs and arms, followed by convulsive movements, occur, and the patient complains of paræsthesia of various forms. In milder cases the symptoms of gastro-intestinal irritation and of muscular weakness attend the fever. In the more severe cases fever is replaced by collapse.

Poisoning by Milk-products.

Gastro-intestinal and choleraic symptoms ensue. Some of the diarrhoeas of infancy are types of this intoxication. The high degree of fever that occurs is well known. In cheese-poisoning the fever is not continuous as in the other forms, the temperature becoming subnormal with the onset of collapse.

Poisoning by Shell-fish.

In mussel-poisoning the symptoms are those of an acute mineral poisoning with profound nervous symptoms. Fever does not attend this condition, but collapse follows quickly. There are no gastro-intestinal symptoms.

Fish-poisoning is also unattended by fever, collapse occurring early.

AFEBRILE INTOXICATIONS.

Herein will not be considered those important *afebrile intoxications* due to disease of the *ductless glands*. The latter include some diseases of the suprarenal bodies (Addison's disease), the thyroid gland (exophthalmic goitre and myxœdema), the lymphatic glands (status lymphaticus), and the spleen, which will be considered in their respective chapters.

Alcoholism.

Acute Alcoholism. In this state the reeling gait, the incoherent speech, followed by narcosis, are generally familiar. The temperature is afebrile. Often, indeed, it is subnormal, and when equal on both sides of the body is very suggestive. The flushed face, possibly slightly dusky, and the injected eye, would lead us to suspect the presence of fever. The odor of the breath furnishes a clue. The heavy breathing, the full pulse, the dilated pupils, the stuporous rather than comatose state, are accompaniments of this intoxication. The flaccid limb of one side would point to hemiplegia from hemorrhage, especially if the coma is deeper than usual and the stupor more marked. But the possibility of *uræmia* and *apoplexy*, or both, occurring in a drunken subject must be borne in mind.

Chronic Alcoholism. When the poison is taken for a long time it acts as a tissue poison and a check upon waste. Epithelial and nerve degeneration and fibrous over-growth follow the first or poisonous irritative action; and fatty change the second. In the alcoholic, tremor of the hands and tongue is seen. The action of the muscles is unsteady. The mind is dull, the temper irritable, forgetfulness is most common, and later a dementia and epilepsy may ensue. Alcoholic neuritis, to be described later, is of frequent occurrence.

Gastro-intestinal catarrh, with poor appetite and constipation, is most liable to ensue, and later cirrhosis of the liver and kidneys; endarteritis, with its train of pathological processes, including myocarditis, and visceral sclerosis also arise.

Grain-poisoning.

Three forms are seen. When the grain is contaminated by ergot, symptoms known as *ergotism* occur. Chronic ergotism may cause gangrene or a train of nervous symptoms in which convulsive movements are most prominent. In the gangrenous form the toes and fingers are the seat of mortification. The process is preceded by anæsthesia, paræsthesia, and pain. In the convulsive form there is slight fever with some weakness

and tingling sensations in the body. Cramps and contractures occur in the extremities, continuing for hours or days, and relapsing frequently. A mild delirium or the development of melancholia or dementia attends the convulsive form.

In other intoxications fever is not so pronounced. In *lathyrism* the symptoms are those of spastic paralysis, which may proceed to paraplegia. In *pellagra*, a disturbance due to maize, there are disorders of digestion, loss of sleep, general pain, and debility. The digestive symptoms are those of salivation, dyspepsia, and diarrhœa. A peculiar erythema arises. Subsequently, desiccation and desquamation of the epidermis occur, and often small boils develop. Headache, backache, spasms, and paralysis of the legs occur in the severe and chronic forms. The nervous symptoms may give way to melancholia.

Lead-poisoning.

Intoxication due to lead, or plumbism, may be acute or chronic. In the *acute* form we have symptoms of gastro-intestinal irritation with constipation and extreme colicky pains. Anæmia may develop rapidly, and pronounced nervous symptoms arise. Among the latter we have neuritis, convulsions, epilepsy, and delirium. Hemorrhages from mucous membranes may be seen, and a form of nephritis develops rapidly. The urine contains albumin and tube-casts. Fever is not a pronounced symptom.

The characteristic symptoms of *chronic* poisoning are :

a. *Saturnine cachexia*, in which anæmia is most pronounced.

b. *Colic*.

c. *Paralysis*, which may be acute, subacute, or chronic, and which usually develops without fever. The paralysis may be antibrachial, causing characteristic wrist-drop ; brachial, in which the scapulohumeral form of paralysis is seen, or of the Aran-Duchenne type, resembling chronic anterior poliomyelitis. Another is the peroneal type, in which the lateral peroneal muscles, the extensor communis of the toes, and the extensor proprius of the big toe are paralyzed, causing the steppage gait. Finally, paralysis of the adductor muscles of the larynx occurs in lead-poisoning. The paralysis often extends from a local group of muscles throughout the body, presenting symptoms like those of an ascending paralysis with rapid wasting. In other instances the general paralysis occurs primarily, the wasting and loss of power going hand in hand. Fever sometimes attends a general paralysis in lead-poisoning.

d. The *cerebral* symptoms of the acute form have been mentioned. In the chronic cases they may also occur.

Optic neuritis, or neuroretinitis, is common. *Delirium*, with hallucination, may occur. *Tremor* is a common symptom. It must not be forgotten that headache, convulsions, epilepsy, and delirium may be manifestations of *lead encephalopathy* even in cases in which the history of exposure to lead is not direct.

e. Chronic lead-poisoning leads to *arterial sclerosis* and *contracted kidneys* with hypertrophy of the heart.

f. *Gout* is very common, and may be seen in both acute and chronic forms, particularly in the big toe.

g. As described in the section in which the mouth and gums are discussed, the *blue line* is the specific symptom of lead-poisoning. The reader is referred to that chapter for a description of the line. It must be remembered that in all forms of obscure nervous disease, in gastro-intestinal irritation, in arteriosclerosis, and gouty arthritis, this line must be looked for.

Arsenic-poisoning.

Acute arsenical poisoning is attended by symptoms of gastro-intestinal irritation followed by the rapid development of collapse. Fever is not a prominent symptom unless recovery is about to take place. The temperature is subnormal, but as the collapse symptoms disappear fever due to gastro-intestinal ulceration develops.

In *chronic* arsenical poisoning the fever occurs only if there is great irritation of the mucous membranes, as of the conjunctiva, mouth, or pharynx. In this form, in addition to the irritation of these mucous membranes, there may be subacute gastro-intestinal catarrh, with diarrhoea. In other instances there is profound anæmia and debility, with paræsthesia and neuralgia. In others, again, paralysis like that of lead-palsy may occur. It must not be forgotten that puffiness under the eyelids may be due to this cause.

CHAPTER III.

CONSTITUTIONAL DISEASES.

Gout.

A DISEASE characterized by specific arthritis, associated with uric acid in the blood and the deposit of sodium urate in the joints, or manifesting itself as a diathesis in which occur other inflammations of non-articular tissues and various disturbances of functions of organs, the blood also containing uric acid.

Gout is common in Europe, particularly in England, but in its articular form is rare in this country. There is an hereditary predisposition in from 50 to 60 per cent. of the cases. It results from over-eating of rich foods and the drinking of malt liquors, associated with insufficient exercise and excretion. Garrod has called attention to its association with lead-poisoning. Paroxysms are induced by indiscretions in eating or drinking, by nervous shock or great mental strain, by exposure to cold or injury, or by over-work and sexual excesses. The characteristic phenomena of gout are preceded for a variable time by acid flatulent dyspepsia, colicky pains in the stomach and bowel, constipation alternating with diarrhoea, and the excretion of scanty, heavily loaded urine. Accompanying these dyspeptic symptoms often are impairment of physical and mental vigor, irritability of temper, and hypochondriasis. In other cases the premonitory symptoms are palpitation of the heart, or dyspnoea resembling asthma, or various nervous symptoms, as drowsiness, insomnia, or headache.

Acute Articular Gout. In acute articular gout the onset is often sudden, especially in the first attack. The patient may go to bed in apparent health, but wake up early in the morning with a feeling of discomfort or uneasiness, usually in the great toe. In some cases the pain is agonizing from the first. The patient finds he is unable to put his foot to the ground without torturing pain. The ball of the great toe is hot, swollen, red, and exquisitely resentful of the slightest touch or jar of the bed. The veins are swollen and the joint stiff. There are slight fever, perhaps chilliness, thirst, coated tongue, constipation; scanty, high-colored urine depositing urates on cooling; the skin is warmer than normal, and there is slight perspiration. The pain usually abates during the day and increases at night. It is aggravated by motion and attended by painful muscular cramps. By the end of the first day or two the swelling increases and the pain lessens, owing to diminished tension of the part. Pain on motion is still great, however, and without treatment may continue for a week or two; under treatment the paroxysm subsides in four or five days. Both great toes may be attacked in the first seizure, more often alternately than simultaneously, and sometimes other joints

than those of the toe are affected. After subsidence of an attack the urine contains a larger quantity of uric acid, and the patient feels better in health and spirits than for some time. A second attack may be postponed for several years, but usually after that the intervals of freedom steadily diminish until an attack recurs every few weeks or months, and the patient may be scarcely ever free from it. Other joints than the toes, particularly those of the fingers, become involved in subsequent attacks.

The Blood. Neusser has attributed to gout and the uric acid diathesis the presence of granules, observed after staining, in the white corpuscles, but they have been found in other affections, and are not diagnostic. The nature of many otherwise obscure gouty manifestations or arthritic changes may be determined by an examination of the blood-serum. Collect the serum which accumulates in a blister and examine for uric acid. (See Blood.)

Chronic gout results from repeated acute attacks. It is characterized by deformity of the affected joints, around which are deposited chalk-stones (tophi) of sodium urate. Similar deposits occur in the helix of the ear. The first appearance is that of a clear vesicle under the skin, which subsequently becomes chalky-white and solid. The deposits of sodium urate occur not only in the cartilages of the joints, but in the ligaments and bursæ also, resulting in great impairment of motion and deformity. "In extreme cases an appearance is presented by the hand very closely resembling a bundle of French carrots with their heads forward, the nails appearing to take the place of the stalks." (Garrod.)

Gouty abscesses consist of collections of liquid and solid sodium urate, which discharge, with or without pus, through the skin. A patient may have a number of them with but slight impairment of the general health. They may even act as a helpful vent to the system.

In so-called *retrocedent gout* the external joint-manifestation is suppressed or replaced by an internal inflammation, as of one of the serous membranes.

Gout attacks the nervous system, causing headache, delirium, and sometimes apoplexy, apoplectiform seizures, epilepsy, mania, various neuralgias, and spinal symptoms. It also affects the heart and blood-vessels, causing valvulitis and chronic arteritis. The symptoms presented by the digestive organs have been mentioned. They are often premonitory of an attack. The kidneys may be affected, causing typical contracted kidney, or there may be chronic cystitis and urethritis.

Rheumatoid Arthritis.

Rheumatoid arthritis, or rheumatic gout, is an affection characterized by acute or chronic inflammation of the joints, of progressive character, and resulting in deformities. It is attended with very little fever, and occurs apart from any known systemic disease.

It may be acute or chronic. The *acute* form differs but little in its manifestations from acute rheumatic fever. Several joints are enlarged, tender, and painful. Constitutional symptoms, such as fever, loss of appetite, frequent pulse, thirst, and furred tongue, occur as in rheumatism.

Profuse acid sweats, however, are absent, and so is the tendency to serous inflammations. Moreover, while the larger joints, as in rheumatism, may be affected, the smaller ones also, especially of the fingers and toes, are inflamed and often the seat of serous effusions. Furthermore, the inflammation persists in the affected joints and does not jump from one to another. Instead of disappearing in a few weeks, it drags on for a much longer time. The pain subsides, but the swelling persists, and permanent deformity results in at least some of the joints. The muscles of the arms and legs waste and are affected with painful spasms. The disease is most common in young women exhausted by repeated pregnancies or prolonged lactation, and is favored by poverty, privation, and cold.

The *chronic* form is much more common. It also attacks most frequently young women who are exhausted or are subjected to great fatigue. There is pain, numbness, or formication in a joint, as the knee. The joint becomes tender, painful, and may be slightly swollen. This subsides after a while, but sooner or later the same joint or another one becomes affected. The process is persistent, one joint after another is attacked, and gradually all the joints may become greatly distorted, enlarged, and the seat of contractions. There may be no impairment of general health, or at most only dyspeptic symptoms. The progress

FIG. 329.



Rheumatic arthritis. (Original.)

is interrupted from time to time by remissions. Pain may be severe and subject to nocturnal exacerbations. The shape is altered by the effusion into the joint and adjacent bursa, by thickening of the tissues around the joint, growths of new bone on the joint-extremity of the bones, absorption of the articular cartilages, and growths of new cartilage in the synovial sheaths. These alterations may be accompanied by relaxation of ligaments, muscular contractures, and luxation of the joints. The joints crack and creak like rusty hinges, are sore and stiff, and the attached muscles are affected with painful cramps. (See Fig. 329.) Great enlargement of the joints at times occurs from the causes already mentioned and from infiltration of the overlying tissues. The enlargement

is rendered more conspicuous by the atrophy of adjacent muscles. (See Fig. 30.) Physicians with large experience in this affection lay stress upon the frequent involvement of the *temporomaxillary articulation*, which is never seen in gout.

In addition to the articular symptoms other phenomena attend the process. One of the more common is increased *frequency* of the *pulse*. Although the patient is afebrile, the average pulse-rate is 100 to 120, or even higher. Moreover, the pulse is soft and compressible, in contradistinction to the pulse of gout and rheumatism. It is worth noting that a return to the normal pulse-frequency is a sign that the progress of the disease is arrested, although the joint-lesions remain.

The condition of the *skin* is characteristic. It is soft and often much freckled, while the complexion is fair. C. T. Griffiths has observed the pigmentary cutaneous changes, along with neural symptoms, prior to the joint-manifestations, and describes two forms: a diffuse melasmic discoloration, and dark-brown spots resembling moles, but not raised. Moisture of the skin with clamminess is common. It is limited to the palms of the hands or may occur in the distribution of certain nerves. The sweats are not acid; they are usually local, but may be profuse. *Pain* independent of the joint-lesion is due to neuritis, and may precede the joint-trouble. It is not confined to the nerve-trunks, but affects the smaller branches which are distributed to muscles, as the group at the base of the thumb. *Numbness* and tingling are often present.

The disease is steadily progressive. In extreme cases not only are the limbs crippled, deformed, and helpless, but there is fixation of the cervical spine and of the articulations of the jaw, so that the patient can not move the head or masticate food. The first phalanx of the fingers is either flexed upon the metacarpus or extended, and the terminal phalanx in like manner is either markedly flexed or extended upon the second, or these two phalanges are held in extension, while the first phalanx is, as usual, decidedly flexed upon the metacarpus. The hand is pronated and the fingers turn toward the ulnar side. (Palmer Howard and Charcot. (See Fig. 329.) The foot is abducted and flattened, and the great toe abducted across and above the other toes. Rarely it may be beneath the other toes. The metatarsophalangeal joint is enlarged.

A variety of the disease is sometimes encountered, chiefly in old persons (senile arthritis), in which the tendency is to involve one or two joints, particularly the hip, or hip and knee. It is of slow progress, and is otherwise attended with the same deformities as the usual polyarticular form.

Diagnosis. Rheumatoid arthritis is distinguished from *gout* by the absence of heredity and by its development under the exhausting influences of repeated pregnancies, lactation, poverty, and malnutrition. Rheumatoid arthritis is progressive, with occasional remissions; gout occurs in successive attacks, with intermissions. Uric acid is absent from the blood in the former and is present in gout. Rheumatoid arthritis in the vast majority of the cases is subacute or chronic. The acute form is distinguished from acute gout by the duration of the paroxysm and the absence of intermissions; by there being less heat, swelling, and redness

of the joints, and less infiltration of the soft parts; by the fact that large and small joints are involved, and that there is no special tendency to inflammation of the great toe.

From *chronic gout* rheumatoid arthritis is distinguished by the absence of hereditary predisposition, of repeated acute attacks, and of the causes of gouty paroxysms—indulgence in sugars, acids, malt liquors, etc. Moreover, rheumatoid arthritis most frequently begins in the hands, and is symmetrical and bilateral. Gout has a predilection for the great toe, and is unilateral. Again, gout attacks well-fed males most frequently after the age of thirty years, while rheumatoid arthritis tends to attack women under the depressing influences already mentioned. It may, however, occur in both sexes, and even be associated with gout.

Rheumatic fever is distinguished from acute rheumatoid arthritis by its tendency to involve the larger joints, its erratic course, acid sweats, and heavy deposits of urates from the urine, its shorter course, its tendency to heart complications, and its subsidence without impairment of the usefulness of the joints.

Chronic articular rheumatism is distinguished by the preceding history, the tendency to seasonal exacerbations, by its involving fewer joints, and not being so symmetrical in the joints affected. It does not produce so great deformity as is common in rheumatoid arthritis, nor is it so likely to affect the vertebræ and jaws. The existence of valvular heart disease or a history of antecedent chorea is in favor of rheumatism.

The joint-affections of *tabes dorsalis* are distinguished by the associated symptoms of inco-ordination and absent knee-jerk, by their sudden onset without pain or fever, by the occurrence of large effusions into the joints, with subsequent disorganization, fractures, and dislocations.

Gonorrhœal arthritis is distinguished by the history of gonorrhœa or the existence of a discharge from the urethra, by the tendency of the disease to attack the larger joints, particularly the knee or shoulder, and to become fixed in one, not wandering from one to another. The affected joint suffers effusion, and the synovial membranes and bursæ are inflamed. The process is very chronic but indolent, and the heart rarely becomes affected.

Chronic Articular Rheumatism.

The patient has pain and stiffness in one or more joints or in the contiguous tissues. The joints most frequently affected are the shoulder and knee. The pain is more or less constant, but worse in damp weather or on the approach of a storm, and worse also at night in many cases. Conversely, it improves in warm, dry weather. There is not much if any tenderness, and rarely any swelling or elevation of temperature. The joints very frequently creak and grate on motion. In the interval between the attacks there is no impairment of the usefulness of the joints. In very chronic cases there may be some atrophy of muscles and permanent stiffness, even fibrous ankylosis. In some cases there are repeated attacks of subacute articular rheumatism, accompanied by the usual symptoms and joint-effusions.

Chronic articular rheumatism is distinguished from *chronic gout* by the

fact that there is no special tendency to involve the great toe, by the absence of the deformities resulting from gout, and the absence of deposits of sodium urate in the ears, fingers, and around the joints.

Muscular Rheumatism.

In this variety of rheumatism there is pain in the affected muscles, which often comes on suddenly in the night, or is first noticed when the patient attempts to rise in the morning. The pain when the patient is at rest may be inconsiderable, rarely amounting to more than a dull, aching, sore feeling; on attempting to move, bend, twist, or straighten himself, however, the patient catches himself suddenly on account of the agonizing tearing or burning pain. When the muscles are relaxed, the patient is fairly comfortable. Sudden movement is the most painful. The affected muscles are tender to the touch and to sharp blows. Muscular rheumatism may be acute or chronic. In the latter the symptoms are very much like those of chronic articular rheumatism, except that the muscles and not the joints are affected. There is the same proneness to recur in unfavorable weather and in cold, damp seasons.

The disease receives different names according to the muscle affected. The most common subvarieties are: *lumbago*, in which the muscles of the small of the back are affected; *pleurodynia*, in which the intercostal muscles suffer; and *torticollis*, in which the sternomastoid and trapezius are painfully contracted.

In *lumbago* the patient holds himself rigid and is unwilling to rotate the trunk upon the vertebræ. Often the most comfortable position is that in which he sits and bends slightly forward over another chair. Motion is painful, but pressure is not. Fever is absent. There is a history of repeated attacks, or of exposure, such as lying upon damp ground. *Lumbago* needs to be distinguished from disease of the spinal membranes, from disease of the vertebræ, aneurism, abdominal abscess, and diseases of the uterus and ovaries. The diagnosis of rheumatism is arrived at by exclusion.

In *pleurodynia* there is usually tenderness upon pressure as well as upon motion and deep inspiration. The pain is of the same sore, burning character, aggravated by coughing and sneezing. The patient breathes as little as possible, and often inclines the body toward the affected side to lessen the motion. *Pleurodynia* is distinguished from pleurisy by the absence of fever, cough, and, above all, of friction-sounds. In intercostal neuralgia there are painful points upon pressure, whereas in *pleurodynia* firm pressure is grateful, though tapping is painful.

In *torticollis* the head is drawn to one side and fixed in that position. The sternomastoid especially is rigid and tender on pinching. In spinal affections the head is retracted, and there are antecedent symptoms, as headache and darting pains with fever.

In rheumatism of the *abdominal muscles* the pain may be so acute as to simulate peritonitis. The surface pain of hysteria which gives rise to so-called "hysterical peritonitis," simulating true peritonitis and rheumatism, may be noted.

Rhachitis.

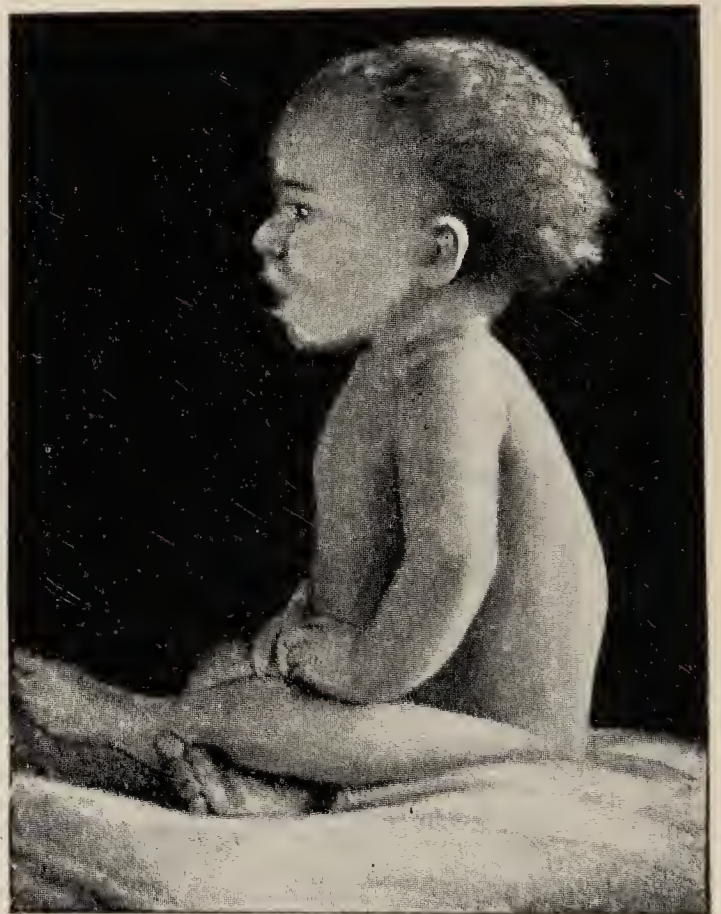
In this affection the size of the body is lessened. For its recognition it is important to know how rapidly the osseous deposits in childhood have formed. The fontanelles and the epiphyses must be examined. If the fontanelles are open after the normal time of closure, or if the epiphyses are enlarged and lack firmness, the condition points either to simple malnutrition or to rhachitis.

FIG. 330.



Rhachitis: attitude in sitting; one hand raised to exhibit swelling at the wrist. (WILLIAMS.)

FIG. 331.



Rhachitis in moderate degree in a boy aged fifteen months; showing backward excurvation of the spine. (WILLIAMS.)

Rhachitis usually develops in childhood, and is most common in children with unfavorable hygienic surroundings, who have lived upon a starchy diet and have taken cows' milk for too long a period of time. A child that has been nursed during the mother's pregnancy is apt to have the disease.

In rhachitis late development of the *teeth* is observed. If the ribs are examined, nodules will be detected at the junction of the bone with the cartilage. These may be seen, as well as felt, if the child is thin. They form the so-called *rhachitic rosary*. The thorax also is changed in shape. At the junction of the cartilages and ribs a depression takes place which is continuous with a groove passing out from the ensiform cartilage toward the axilla. This transverse curve is known as *Harrison's groove*. It may deepen with inspiration. The sternum projects, forming the so-called "pigeon-breast." (See Thorax.) Such deformity must not be confounded with a similar one seen in adenoid disease. Changes at the lower end of the radius and ulna, and sometimes at the end of the humerus, are noticed. The parts are enlarged at the junction of the

shaft and epiphyses. There may be thickening of the clavicles at the sternal ends. In the legs the lower end of the tibia becomes enlarged, and at times the upper end, or even the shaft, becomes thickened. The child becomes bow-legged, or the tibiæ and femora may arch forward. Knock-knee sometimes occurs. The bones of the vertebral column and of the pelvis are also affected. The spine is usually kyphotic, and sometimes there is lateral curvature as well. The contraction of the pelvis is such as to narrow its outlet—a matter of much importance for the future of females.

The child's head is quite characteristic. It has been mentioned that the fontanelles remain open for a long time, and areas of ossification are imperfect, so that the bone yields to the pressure of the finger. This occurs particularly at the side, and the term *craniotabes* is applied to it. The large head is square in shape, not globular, when seen from above downward. The appearance of the face is peculiar: it is proportionately very small, especially in the lower two-thirds, while the forehead is broad and square. In addition to changes in the bones a child presents other evidences of defective nutrition, such as marked pallor, flaccidity of the muscles, and general muscular weakness which results in an inaction resembling paralysis.

The disease usually progresses slowly, and is eminently chronic. A form is seen, however, in which the course is more acute. With some gastro-intestinal disturbances there are mild fever, considerable weakness, and great restlessness. Sleep is disturbed, and pain is complained of, if the child is of an age to make such complaint. *Soreness* of the body is observed on handling the child; and of its own accord, on account of the pain and soreness, it avoids all customary movements. The child lies on its back and shrinks from attempts to disturb it. The pain is not only caused by handling of the muscles, but the bones also are sore and tender. Sometimes the most marked manifestations of the more acute forms are the gastro-intestinal symptoms. It may often happen that vomiting and diarrhœa have this rhachitic condition as an underlying basis.

With the above symptoms, *perspirations* about the head are common, and occur also in chronic cases. There is usually more heat of the head than is natural, hence in sleep the child rolls the head. This rolling causes the hair on the back of the head to wear off, a characteristic of rhachitis when observed along with changes in the skeleton.

In both the acute and the chronic forms *enlargement of the liver and spleen* is observed. The enlargement is not only actual, but a false enlargement may also be seen from distortion of the organs on account of changes in the vertebræ and ribs. The abdomen is prominent, usually on account of flatulence, although the enlarged organs contribute to the swelling.

Nervous phenomena are common in the course of rhachitis. *Tetany* limited to the upper extremities, and *laryngismus stridulus* are the most frequent. Either of these complications may occur before the disease is otherwise suspected.

Diagnosis. The possible presence of rhachitis must not be overlooked

in cases of chronic vomiting in childhood. The acute form of the disease must not be confounded with scurvy, as often happens in the case of children. It must not be forgotten that scurvy may set in in the course of rhachitis. In scurvy the pain, tenderness, and weakness are limited to the lower extremities. The immobility of the extremities may go on to pseudoparalysis. The tenderness, however, is great; œdema is more pronounced, and local areas of periostitis are more common. In scurvy the gums are swollen and may be spongy or the seat of ecchymoses. The most decisive diagnostic criterion is the therapeutic test, scurvy rapidly yielding to a proper regimen.

Diabetes Insipidus.

This form of diabetes differs from the saccharine in that the urine is normal except for its low specific gravity, from 1001 to 1005. The disease may come on suddenly after mental emotion or develop gradually. The amount of urine may range from 10 to 40 pints. It is pale and watery. The solid constituents are not reduced. Urea is sometimes increased, but abnormal constituents are rare. The passage of large amounts of urine induces thirst, but otherwise the symptoms do not tally with the symptoms of diabetes mellitus. The patients are usually well nourished.

The disease is usually secondary to some organic disease of the brain, or of the abdomen, as tuberculous peritonitis, abdominal tumors, or aneurism. It usually occurs in males, and is often hereditary. It is most common in young people. Traumatism, meningitis, affections of the brain involving the sixth nerve, tumors of the brain or of the medulla are causal factors. It may follow fright, a protracted debauch, or perturbation of the nervous system from other causes.

Diagnosis. The diagnosis is not difficult. It must be distinguished from the polyuria that is seen in chronic interstitial nephritis and in amyloid disease. In hysteria, polyuria is common, although transitory. The presence of the stigmata and other hysterical manifestations establishes the diagnosis in hysteria.

Diabetes Mellitus.

The occurrence of any of the following conditions should lead to an examination of the urine for *sugar*, and an estimation of the quantity of urine passed in twenty-four hours, apart from the routine examination, which should be made in every case of chronic disease or of obscure acute disease.

1. *Muscular weakness without cause*, progressive and rapidly advancing to an extreme degree.

2. *Emaciation*. In young subjects this is rapid in cases of diabetes. In older patients it is not so striking, particularly if the gouty diathesis is present.

3. *Thirst*. This is of common occurrence in diabetes. If the amount of fluids taken be compared with the amount of urine excreted, it will

be found that the two bear a definite ratio. The thirst is greater immediately after meals, although the patient does not necessarily have indigestion.

4. *Hunger.* Excess of appetite, boulimia or polyphagia, also occurs in diabetes. The amount of food that is taken is sometimes enormous, and the ravenous manner in which it is devoured is revolting.

5. *Loss of sexual power.*

The five symptoms just mentioned, with increased frequency of micturition, are the common symptoms of diabetes mellitus. They may develop gradually. In rare instances the onset is sudden. The occurrence of these symptoms should lead at once to an examination of the renal secretion.

Three special characteristics of the urine are observed : (a) The *quantity* is increased, so that from 6 to 10 pints, or even as much as 30 to 40 pints are passed in twenty-four hours. (b) The *specific gravity* ranges from 1025 to 1045, and may even be higher. (c) The presence of *sugar*. The sugar is detected by the ordinary tests. (See Examination of Urine.) In addition the urine is usually of a pale color, of a sweetish odor and acid reaction.

In addition to thirst and increased appetite, some gastro-intestinal symptoms may be of diagnostic importance. Of these, first, the appearance of the *tongue* is characteristic. It is dry, red, and glazed. The dryness is increased because of the scanty flow of *saliva*. The *gums* are swollen and spongy, and marginal gingivitis and *stomatitis* are often present. There are no marked dyspeptic symptoms. *Constipation* is of common occurrence. In diabetes the other secretions of the body are diminished. *Perspiration* does not occur excepting in inflammatory complications. The *skin* is harsh and dry. As the disease progresses, the *heart's* action becomes weak and the *pulse* frequent with higher tension. The *temperature* of the body is usually below normal.

Diabetes may occur at any age, but is most frequent in adult life. In young adults the symptoms are more pronounced, and the duration shorter. In patients past middle life the disease may continue for a number of years without marked interference with the health and nutrition.

While the symptoms just mentioned should lead to an examination of the urine, diabetes mellitus may not be indicated by any of the usual objective or subjective symptoms. It may happen that none of these symptoms is sufficiently marked, and that only by routine examination of the urine, or by the occurrence of affections known to be associated with sugar in the urine, is the disease discovered.

Complications. Of the complications which should lead to the suspicion of sugar in the urine the following are the most important :

1. **Cutaneous Complications.** Boils and carbuncles should always lead to an examination of the urine. Pruritus and chronic eczema may have diabetes in the background. Gangrene of the extremities, chiefly of the feet and legs, and gangrene in other situations, are of common occurrence in the course of diabetes.

2. **Lung Complications.** Tuberculosis of both the chronic and the acute

pneumonic type is frequently associated with diabetes. Lobar pneumonia is apt to occur. In all cases of pneumonia the urine should be examined for sugar. Its presence would modify the prognosis of an otherwise favorable case. Gangrene is likely to ensue in acute and chronic lung affections. Gangrene of the lung in the course of diabetes may be latent and recognized only by the odor and the character of the expectoration, or it may run an acute febrile course.

3. Nervous Symptoms. *Diabetic coma* may develop in the course of the disease. In young subjects, particularly, the occurrence of coma should lead to a suspicion of diabetes. It may occur before the disease has been recognized. Coma may occur without any premonitory symptoms whatsoever, the patient reeling for a short time, and complaining of pain in the head as after a debauch. The attack may begin with fainting and prostration, followed by stupor which deepens into complete unconsciousness. It may be preceded by nausea and vomiting or by the lung complications previously mentioned. Extreme dyspnœa, agitation, pain in the head, and some delirium precede the loss of consciousness. The pulse becomes rapid and feeble. For this form of coma the term *acetonæmia* is used. The breath is of a peculiar sweetish odor, due to acetone, and this compound is detected in the urine.

Peripheral neuritis should always lead to an examination of the urine. It may be limited to one group of nerves, or may be more or less general, with symptoms like those of locomotor ataxia, as the lightning-pains, abolition of reflexes, and loss of power in the extensor muscles. Diabetic patients are also subject to neuralgia and to peripheral hyperæsthesia and paræsthesia, probably due to neuritis. The neuritis may be so extreme as to lead to paraplegia.

Eye Symptoms. A curious symptom of diabetes is the occurrence of *cataract*. This may develop at any age, and is often rapid in its course. Cataract or alterations of vision always demand an examination of the urine. Diabetic *retinitis* is sometimes present. Atrophy of the optic nerves or muscular insufficiencies may take place, the latter causing pronounced symptoms of eye-strain.

Aural Symptoms. Ringing in the ears, deafness, the occurrence of acute otitis, are phenomena which arise in the course of diabetes.

Diagnosis. Sugar in the urine occurs temporarily when there is an excess of saccharine diet, or when there is functional disorder of the liver. The sugar is small in amount, and the glycosuria is transient. The diagnosis of true diabetes is not difficult, although the disease may be overlooked unless the habit, previously insisted upon, of invariable urinary examinations is fully developed.

CHAPTER IV.

DISEASES OF THE BLOOD AND OF THE DUCTLESS GLANDS.

DISEASES OF THE BLOOD.

THE blood is a tissue, the origin, growth, and decay of the elements of which have been the source of the greatest interest. In days gone by it was the tissue held responsible for many diseases of unknown origin, so that skin eruptions, scrofula, and other affections were known as "blood diseases." At present we hold only such affections blood diseases as show a demonstrable change in the physical or morphological characteristics of the blood. There is either diminution of the red cells, increase or diminution of the white cells, or diminution of the hæmoglobin. Strictly speaking, most of the blood diseases now so called are really diseases of the blood-making organs—the lymphatic glands or the spleen. It is interesting to note that as late as 1866, J. Hughes Bennett included under diseases of the blood leucocythæmia, chlorosis and anæmia, diabetes, the infectious diseases, rheumatism, gout, and scurvy. The most recent textbook divides the blood diseases into *anæmia*, with two subdivisions, and *leukæmia*. Of course, no one thinks of considering the infectious diseases blood diseases, any more than we think of considering typhoid fever an ulceration of the intestine.

Although the blood diseases are thus limited, it is none the less true that the blood may be the only tissue by an examination of which we can determine the ailment from which the patient suffers. As has been previously related, many infections are recognized in this manner only.

The symptoms of blood affections are due to the physical change in the blood and the effect of this altered blood upon the function or the nutrition of the organs. Many *functional symptoms* thus arising may be the first indications of blood disease, as vertigo, headache, dyspnœa, or palpitation, all very common symptoms. The symptoms may be subjective or objective, or both. The recognition of the former comes from the history of the disease and the complaints of the patient. The latter, or the objective symptoms, are determined by the physical examination of the patient and the examination of the blood.

We recognize scarcely any condition at the present day due to an increase of the bulk of the blood or of the red cells. Plethora is hardly a clinical entity. The symptoms of blood diseases therefore are the symptoms of *anæmia*. In like manner, all the data obtained by inquiry are those which belong to some form of anæmia.

Anæmia.

Anæmia is a condition characterized by a reduction in the number of red blood-cells, or of their hæmoglobin, or of the albumin, or of all

combined. In general we say an anæmia is (*A*) primary, essential, or idiopathic ; or (*B*) secondary, according to (*a*) the absence of a well-defined cause or (*b*) the presence of some recognizable blood-destroying condition. The anæmias may be suitably subdivided as follows :

ANÆMIA	{	Primary.	{	Chlorosis.
				Pernicious anæmia.
{		Secondary.		

The most casual observation may be sufficient for the recognition of anæmia. The color of the surface, the appearance of the mucous membranes, and the evident breathlessness of the patient are indications of diminution in the amount of blood, or of some of its constituents, as the red cells, or of the coloring-matter of these cells. On inquiry it is found that the patient is easily prostrated, that there is breathlessness on exertion (aggravated on ascending any height), that there is palpitation and perhaps cardiac oppression. The patient complains of neuralgias in various parts of the body, and especially of the neuralgia so often seen in the inframammary region of the left side. (See Pain.) Headache is a more or less constant symptom, with the peculiarity that it is increased when the patient ascends stairs, and is often throbbing or pulsating. The anæmic subject has usually a poor appetite and suffers from gastralgia, although it must be remembered that the gastric symptoms of anæmia are as often primary as secondary. Many of the symptoms which attend neurasthenia occur in the course of anæmia.

On physical examination of the patient the appearances above indicated are observed, although grave anæmias may be present, and yet the lips be bright red, the color under the nails fair, and the cheeks flushed, especially if the examination is made in the evening. Reference must be made to the chapter on the Color or Hue of the Surface for a description of the appearance in anæmia.

A study of the heart and bloodvessels usually yields the physical signs that attend anæmia. The vascular phenomena are described in the section on Diseases of the Heart. Here again it must be remembered that considerable anæmia may be present without murmurs in the bloodvessels.

The Blood. The final diagnosis rests upon an examination of the blood. Sometimes the most apparently anæmic subjects yield normal results in blood examination, while the most plethoric in appearance may be very anæmic. The various forms of anæmia give rise to blood-changes in a measure peculiar to the respective variety. The primary anæmias, or hæmolytic varieties, to which pernicious anæmia and chlorosis belong, have characteristics which will be described in the special sections.

In anæmia from hemorrhage the red corpuscles may be reduced to 1,500,000, while the hæmoglobin is reduced to a degree greater than that of the red cells. The leucocytes are increased in number, the polynuclear forms being relatively much more numerous than the other varieties.

The red corpuscles are paler than normal ; their white centres are increased in size. This is known as *achromia*. There is some poikilocytosis. An excess of nucleated red corpuscles, or *blasts*, is seen in grave anæmias. If the *normoblasts* are in excess, active regeneration is in

progress ; if the megaloblasts, there is reversion to embryonal regeneration, a serious import in a case of anæmia. A megaloblast-anæmia is associated with general increase in size of the red cells and an increase of the macrocytes. In fatal anæmia, as in purpura, the red cells are like those in the form just described, although nucleated red corpuscles are absent. The white cells are sometimes reduced in number ; the mononuclear forms may be relatively numerous.

In the oligocythæmic forms of anæmia other than the hemorrhagic, the occurrence of poikilocytosis is constant and marked. Nucleated red corpuscles are not common, although a few of some kind are seen in the severe forms, but large nucleated cells with karyokinetic figures occur. These corpuscles have pale-staining nuclei. Achromic forms, polychromatophiles, and degenerate forms are seen. There is usually moderate leucocytosis in secondary anæmias with increase in the polymorphonuclear elements.

For clinical purposes it is necessary to make a number of divisions of anæmia, though on ætiological and pathological grounds many of them will no doubt soon be grouped together.

I. Toxic Anæmias. The poison may be developed in the body or it may be introduced from without. Toxæmia is, sometimes at least, a factor in the anæmias which develop in the course of acute infectious diseases or during convalescence from them. According to Hunter, pernicious anæmia should be classed under this head. The metallic poisons, particularly lead, mercury, arsenic, phosphorus, the potassium salts, especially the chlorate ; certain of the antipyretics, notably pyrocin ; and the aniline preparations, are capable of producing anæmia.

II. Parasitic Anæmias. Anæmia may be *parasitic*. 1. To this class belongs the anæmia of malaria, which is due to *Plasmodium malarie* described by Laveran, and severe pernicious anæmia caused by *Uncinaria duodenalis*.

2. Certain intestinal worms are found associated with marked anæmias:

a. Bothriocephalus latus sometimes produces a disease closely resembling pernicious anæmia, but whether by direct destruction of the blood, or by the development of toxic products themselves destructive, is uncertain ; the worms may be present in large numbers without giving rise to anæmia.

b. Uncinaria duodenalis is the cause of the anæmia known variously as Egyptian or African chlorosis, tropical anæmia, brick-burners' anæmia, and the anæmia widespread in the Carolinas, Texas, and other parts of this country.

c. Anguillula intestinalis is the cause of "Cochin-China diarrhœa" and its associated anæmia.

3. *Filaria sanguinis hominis* may produce anæmia by blocking the lymph-channels.

4. *Bilharzia hæmatobia* may produce anæmia by inducing hæmaturia.

III. Anæmia from Hemorrhage. Anæmia may be due to *hemorrhage*. In addition to accidental and *post-partum* causes, purpura, hæmophilia, menorrhagia, and metrorrhagia are frequent causes.

IV. Anæmia from Constitutional and Local Diseases. Anæmia is

often a marked symptom of *constitutional* and *local diseases*, such as tuberculosis, syphilis, cancer, rheumatism, scrofula, scurvy, rickets, Bright's disease, chronic catarrhal gastritis, and others. The anæmia here may be due to the malnutrition and interference with digestion brought about by the disease, or, as in the case of Bright's disease, in part to the direct loss of albumin, and in dyspeptic conditions to inability to take and assimilate food.

In many cases of simple symptomatic anæmia the spleen may become progressively enlarged, probably secondarily. In some cases of Hodgkin's disease the spleen is enlarged. It is not fully determined whether there is a primary splenic anæmia.

V. Anæmia of Malnutrition. Anæmia may also be the result of malnutrition from deficient or improper food, or from the poisonous influences of unsanitary surroundings.

I. Primary Anæmia—Chlorosis.

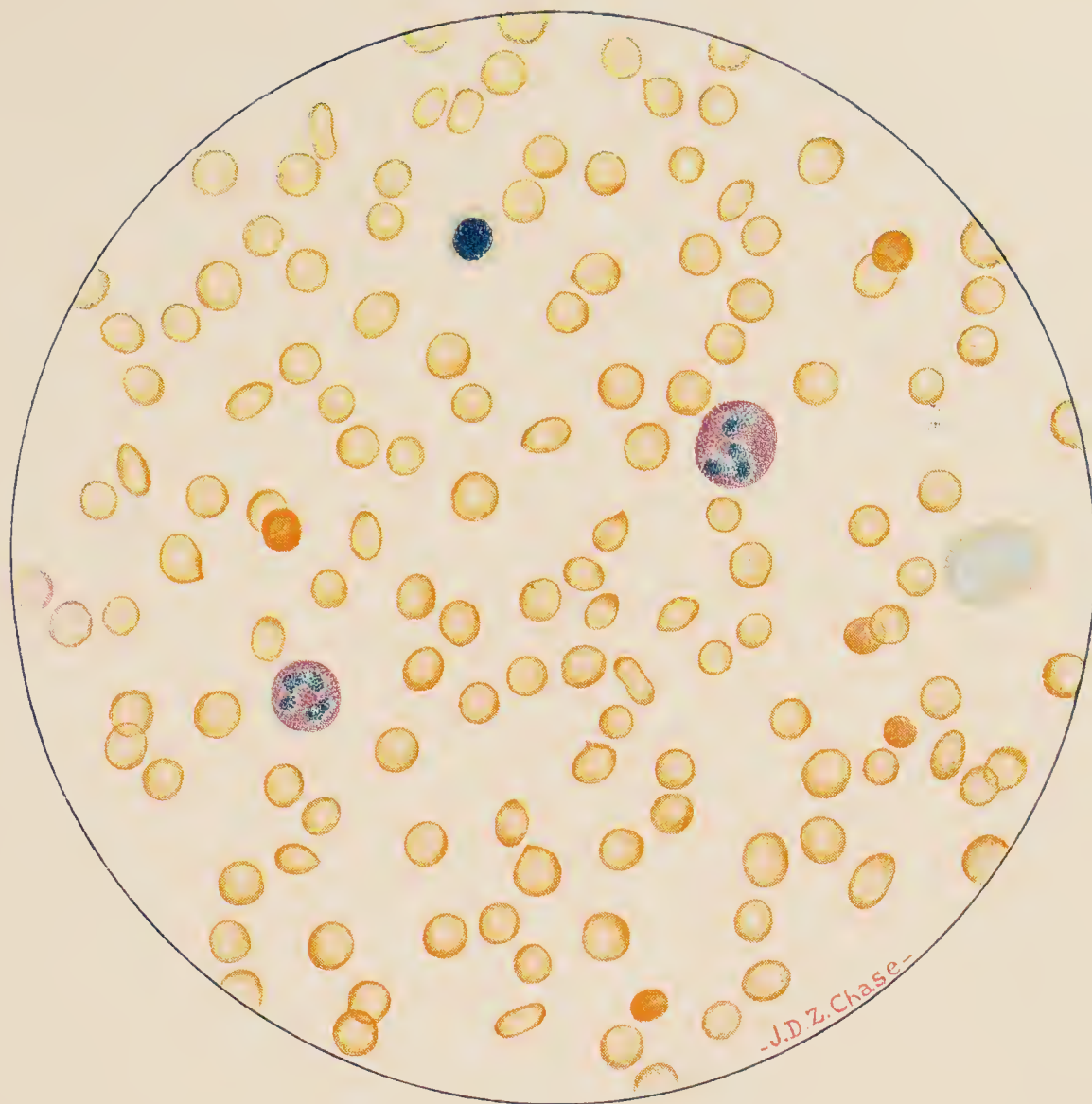
Chlorosis, or chloro-anæmia, is a form of anæmia occurring especially in young girls about the period of puberty and characterized by great pallor with a greenish tint of the skin and mucous membranes, a pearly eye, languor, weariness, suppression or irregularity of menstruation, venous hum in the vessels, dyspnœa, palpitation, dizziness, neuralgias, and an unstable condition of the nervous system. In spite of the extreme pallor there is usually but little loss of flesh. The skin may be pigmented, especially around joints. The bowels are usually constipated; the urine abundant, pale, and of low specific gravity. The digestion is disturbed, the appetite capricious, and the patients sometimes crave unwholesome things, such as earth, slate-pencils, vinegar, and the like. Hyperacidity of gastric juice is commonly present. A systolic murmur over the base of the heart is common. Gastralgia is more common than in other forms of anæmia.

The changes in the blood are very important. There is always a marked reduction in the hæmoglobin, the percentage falling sometimes to 30 or 25 per cent. of the normal. The red blood-cells are usually also reduced, but not in the same proportion as the hæmoglobin. For example, there may be 4,000,000 red cells, but only 30 per cent. of hæmoglobin. Sometimes there is no diminution in the number of red cells; the latter, however, appear pale (achromia), vary considerably in size, microcytes and occasionally poikilocytes are present, and, in severe cases, nucleated red corpuscles are found; occasionally macrocytes occur, but in general the size of the red cells is below that which is usually found. The number of leucocytes varies but little from the normal, but there may be a slight increase. Occasionally there is a rise of temperature, but it is probably due to some complication. (See Plate XVIII.—B, Fig. 1.)

The cause of chlorosis has not been determined satisfactorily. Virchow has established the existence of congenital narrowing of the blood-vessels. Sir Andrew Clark thinks it is due to the absorption of poisonous matter from the intestines; the great benefit that follows saline purgatives in many cases indicates that fecal toxæmia is a factor in these cases.

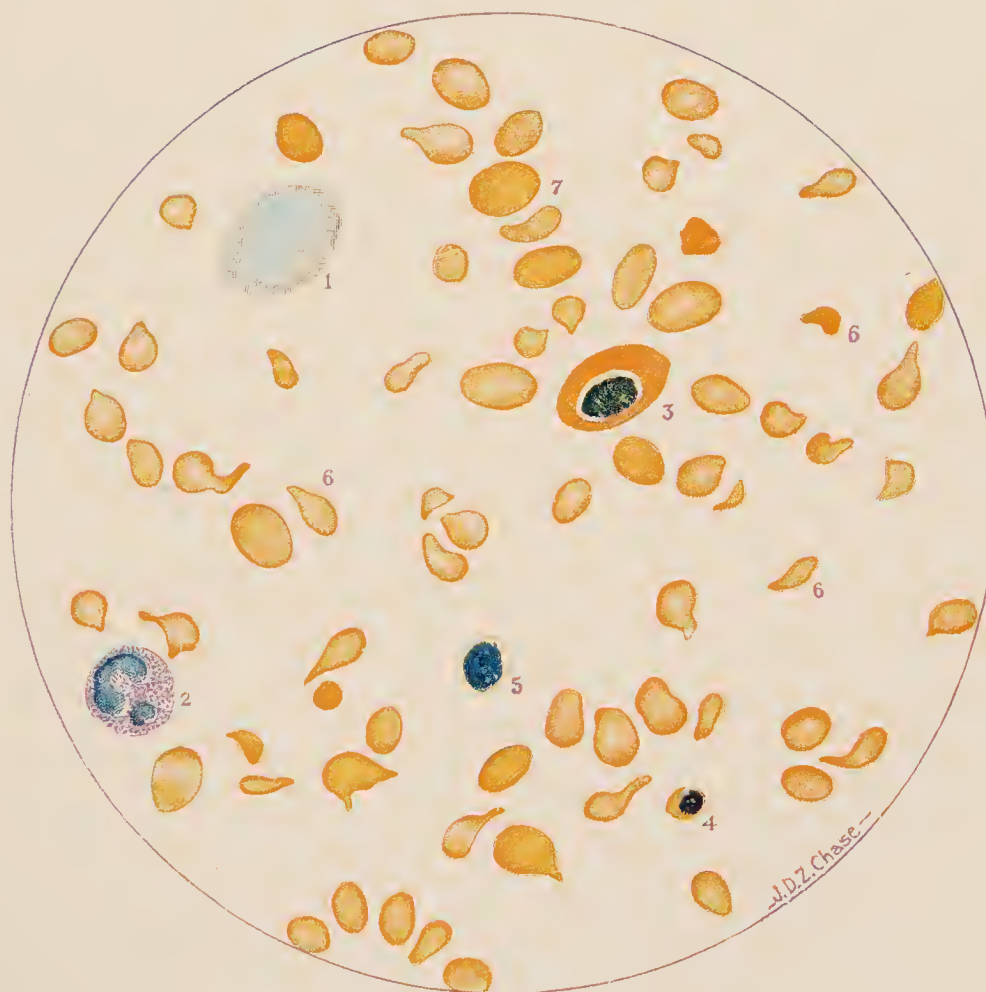
PLATE XVIII-b.

FIG. 1



Chlorosis.

FIG. 2.



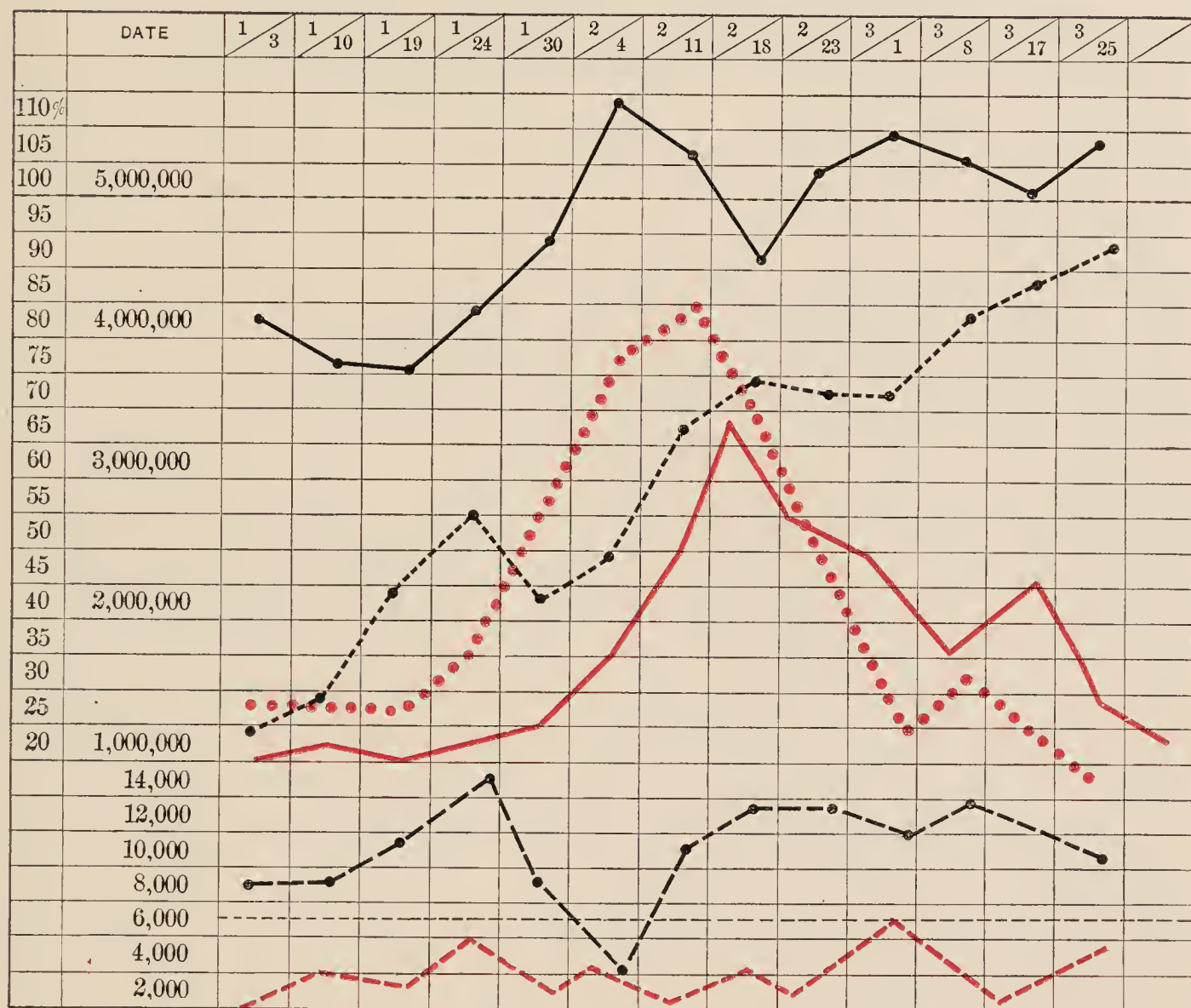
Pernicious Anæmia.

- | | |
|--|--------------------------|
| 1. Large Mononuclear Lymphocyte. | 5. Small Lymphocyte. |
| 2. Polymorphonuclear Leucocyte or Neutrophile. | 6. Poikilocyte. |
| 3. Megaloblast | 7. Normal Red Corpuscle. |
| 4. Microblast | |
- } Nucleated Red Corpuscles.

Forchheimer¹ also looks upon it as intestinal in origin. Sex and puberty are predisposing causes; but chlorosis may occur in boys, and appear in girls before puberty, and in young women considerably after that period.

FIG. 332.

Blood-charts comparing pernicious anæmia and chlorosis.



Chlorosis. Showing improvement after the third week. Continuous lines, number of red cells; small dots, percentage of hæmoglobin; large dots, number of white cells; red lines, pernicious anæmia.

The prognosis is favorable; the disease may, however, be complicated with gastric ulcer, chorea, tuberculosis, and endocarditis. Recovery is often slow and interrupted by relapses.

II. Pernicious Anæmia.

Pernicious or idiopathic anæmia is a form in which the diminution of red blood-cells reaches an extreme degree. It occurs without adequate known cause, and runs a progressive course with remissions; it usually terminates in death.

The disease usually develops slowly and insidiously, the patient presenting the ordinary symptoms of anæmia—pallor, weakness, shortness of breath, palpitation, venous murmurs, loss of appetite, and impaired

¹ Transactions of Association of American Physicians, 1893.

digestion. As the disease progresses the skin becomes of a pale lemon hue, weakness and dyspnœa increase, the patient has attacks of dizziness, faintness, and ringing in the ears; there may be slight œdema; hemorrhages occur from the nose, the bowels, and into the retina. The hemorrhages are small and distinct in the skin and mucous membranes. The urine is of low specific gravity, and usually contains an increased amount of uric acid. According to Hunter, the urine should be dark and contain a pathological amount of urobilin, some renal epithelium, a few casts containing blood-pigment, and an increased amount of iron. The bowels may be disturbed by diarrhœa. In spite of extreme exhaustion, anæmia, and widespread functional disturbance, there is no emaciation; the patient appears well nourished.

Fever. A peculiarity of the disease is the occurrence of fever of an irregular type. The temperature rarely rises higher than 102° or 103° F. in the evenings, and is followed by a morning remission. It is not usually present in the early stages of the disease, may be absent for weeks at a time when the disease is fully developed, and may cease entirely in the later stages.¹

Blood. The *blood* appears pale and watery to the naked eye. There may be difficulty in obtaining by puncture a sufficiently large drop for examination. The specific gravity is lowered, often being 1028 instead of 1055. It has been found deficient in fibrin, iron, and nitrogen.

The blood-changes in idiopathic anæmia are characteristic, and are essential to the diagnosis of the disease. In brief they are (1) very great reduction in the number of red blood-cells; (2) an absolute diminution in the amount of hæmoglobin, but as compared with the number of red cells sometimes a proportionate increase; (3) considerable variation in the size of the cells, the average size of the cells probably being larger; (4) poikilocytosis; (5) nucleated red blood-cells; (6) degenerative cells. (See Plate XVIII.—B, Fig. 2.)

Reduction in the number of red blood-cells (oligocythæmia) reaches a more extreme degree in pernicious anæmia than in any other disease; the number often falls below 1,000,000, and in one case reported by Quincke² the number was only 143,000 per cubic millimetre. The shape of many of the cells is altered; they are oval, elongated, bent, or have projections of their substance (poikilocytosis). The size of the cells varies; there are microcytes and megaloblasts; but the occurrence of a distinct proportion of large nucleated red blood-cells (megaloblasts) is regarded by Ehrlich as almost diagnostic. The average size of the red cells seems to be increased, and so is the proportionate amount of hæmoglobin in each cell. The latter is a very characteristic symptom (the only one, according to Hunter). There are also red corpuscles which are stained slightly and uniformly by basic stains, "polychromatophilia," or which may show punctate stained areas—"granular degeneration"; these are regarded as degenerative by Ehrlich. The leucocytes are "usually diminished in number, showing a relative increase in the small mononuclear elements

¹ See "Idiopathic Anæmia: A Report of Three Cases." Musser, Philadelphia County Med. Soc. Trans., 1885.

² Deut. Arch. für klin. Med., Bd. xx.

(lymphocytes, small transparent forms), while the multinuclear elements are relatively diminished, sometimes being under 50 per cent.”¹

The blood condition is not constant, but is subject to wide variations. Von Noorden has found that in a very short time a change in the form of the blood, a “formal” crisis, may occur. A “formal” flooding of the blood with polynuclear leucocytes and nucleated red blood-cells takes place before a period of improvement; whereas, before a change for the worse and before the final stage the blood becomes poor in leucocytes and nucleated red blood-cells.²

Secondary sclerotic changes in the spinal cord cause late symptoms of *locomotor ataxia*.

The *ætiology* of the disease has not been determined satisfactorily. It is more common in Germany and Switzerland than in other parts of Europe or in America. It occurs most frequently after the twentieth year, and between that and the age of fifty. Excluding the influence of pregnancy and parturition, sex makes no difference. Previous exhausting disease, chronic gastric and intestinal catarrh, great physical over-exertion, exposure, great shock or fright, precede in certain cases the development of the disease. It is probably due to faulty hæmatogenesis and hæmolysis.

Petrone and Halst regard the disease as infectious and its germ identical with that found by Frankenhauser. Von Jaksch supposes that it is brought about by a living contagium. Hunter traces the cause to a poison produced by bacteria in the gastro-intestinal canal.

Diagnosis. The most important diagnostic features of the disease are extreme oligocythæmia, relatively high percentage of hæmoglobin (color-index high), great poikilocytosis, which may, however, occur in any severe anæmia, a noticeable number of large nucleated red blood-cells (gigantoblasts), an average increase in the size of the cells, and all this without emaciation or discoverable local disease that might bear a causative relation to the anæmia. In addition, retinal, subcutaneous, and submucous hemorrhages, a urine with high specific gravity, high color, with urobilin in excess, alternating with urine of low specific gravity, in the absence of organic disease, point to *pernicious* or *idiopathic* anæmia. The distinction from secondary anæmias can not be based on the blood examination alone. The history and the physical signs must play a prominent part, while all possible causes of secondary anæmia must be excluded.

The disease is not so rare as we have been led to believe. The writer has seen a large number of cases in consultation, usually believed by the attending physician to be carcinoma of the stomach or liver, heart disease, renal disease, typhoid fever, or tuberculosis.

Leucocythæmia.

Leucocythæmia, or leukæmia, is a disease of the blood-making organs, characterized by great and persistent increase in the white blood-corpuscles; by diminution in the number of red blood-cells, which are altered in shape and size and display nucleated and degenerate forms; by a lessened amount of hæmoglobin, and by changes in the spleen, lymphatic glands,

¹ W. S. Thayer, Boston Med. and Surg. Journ., February 16 and 23, 1893.

² Quoted by Weiss, Diagnostisches Lexikon.

or medulla of bone. As a rule, the blood in leukæmia presents one of two distinct types: (1) the *myeloid*, associated with great enlargement of the spleen, marked marrow-changes, and little or no enlargement of other lymphatic tissue, usually a very chronic type; (2) the *lymphoid*, in which some one set of lymphatic glands is enlarged, and the enlargement of the spleen sometimes equals that of the myeloid form. These cases may be acute or chronic. The disease occurs twice as frequently in men as in women, and two-thirds of the cases appear between the twentieth and fiftieth years. In women, pregnancy, parturition, and the cessation of menstruation are causative factors, while in both sexes depressing influences upon the body or mind and antecedent disease, particularly malarial fever, have a distinct influence. The first symptom generally noted is enlargement of the abdomen; subsequently the patient complains of pains in the splenic region, weakness, dyspnœa, hemorrhage, œdema, and digestive derangements. Occasionally profuse hemorrhage from trifling cause, as the drawing of a tooth, has been the earliest symptom noted. The increase of white cells and diminution of red cells are progressive, and in the lymphoid form soon become manifest in the pallor of the skin and mucous membranes, and in increasing weakness and dyspnœa. Pallor is not a constant symptom of leukæmia; the myeloid form and a high grade of color are quite consistent.

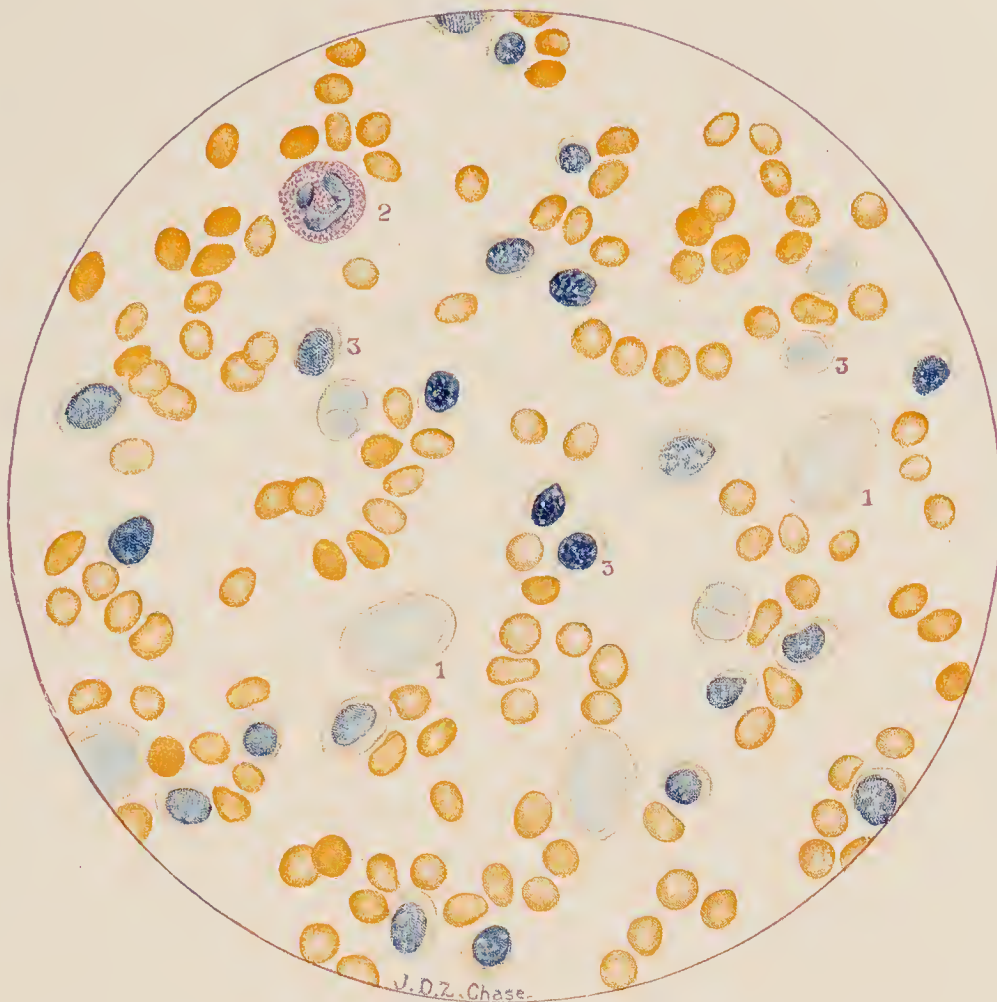
In the so-called *splenomedullary* or *myeloid form* of the disease, myelogenic leukæmia, the spleen steadily enlarges, but may attain considerable size before the patient becomes aware of it. The enlargement is not usually painful, but gives rise to a feeling of distention, weight, and dragging. There may be tenderness on palpation and pressure, and sometimes the patient complains of sharp, stabbing pains, due either to attacks of local peritonitis or to sudden enlargement of the spleen and consequent stretching of the capsule. The splenic enlargement is uniform, so that its shape and characteristic notch are unchanged. Moreover, the spleen remains in contact with the abdominal walls, lying in front of the splenic flexure of the colon, pushing aside the descending colon and small intestine, moving with respiration, and presenting the usual physical signs of a solid organ. Not infrequently the enlargement is so great as to fill the left hypochondriac and iliac regions, and reach beyond the middle line toward the right groin. Sometimes a venous hum can be heard over it. As the result of this enlargement the diaphragm is pushed upward, increasing the dyspnœa already caused by anæmia, and sometimes inducing palpitation. The gastric functions are disturbed from pressure; vomiting and other symptoms of dyspepsia are common.

A rise in temperature is a very common symptom. The fever is of irregular type, usually with nocturnal exacerbations, the temperature not often rising above 102° F. The febrile type may be intermittent or remittent, and sometimes there are periods of apyrexia. The pyrexia is said to be most marked toward the close of the disease. Gowers states that the cases in which there is most fever run a usually rapid course, marked by considerable dropsy and extensive hemorrhage.

As the disease progresses weakness increases; anæmia becomes more intense; œdema, ascites, or hydrothorax occurs; hemorrhages from the

PLATE XVIII-c.

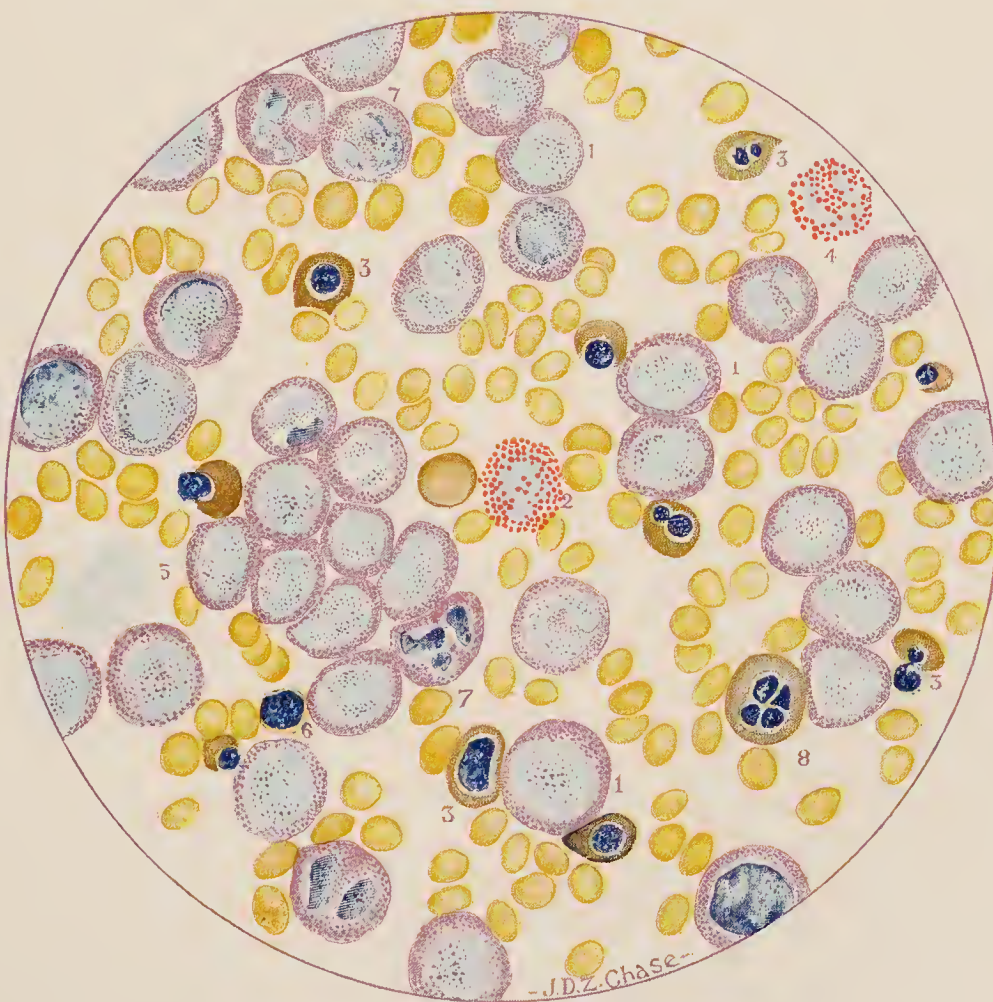
FIG. 1.



Lymphatic Leukæmia.

1. Large Mono-nuclear Lymphocyte. 2. Polymorphonuclear Leucocyte or Neutrophile.
3. Small Lymphocyte, dividing Nuclei.

FIG. 2.



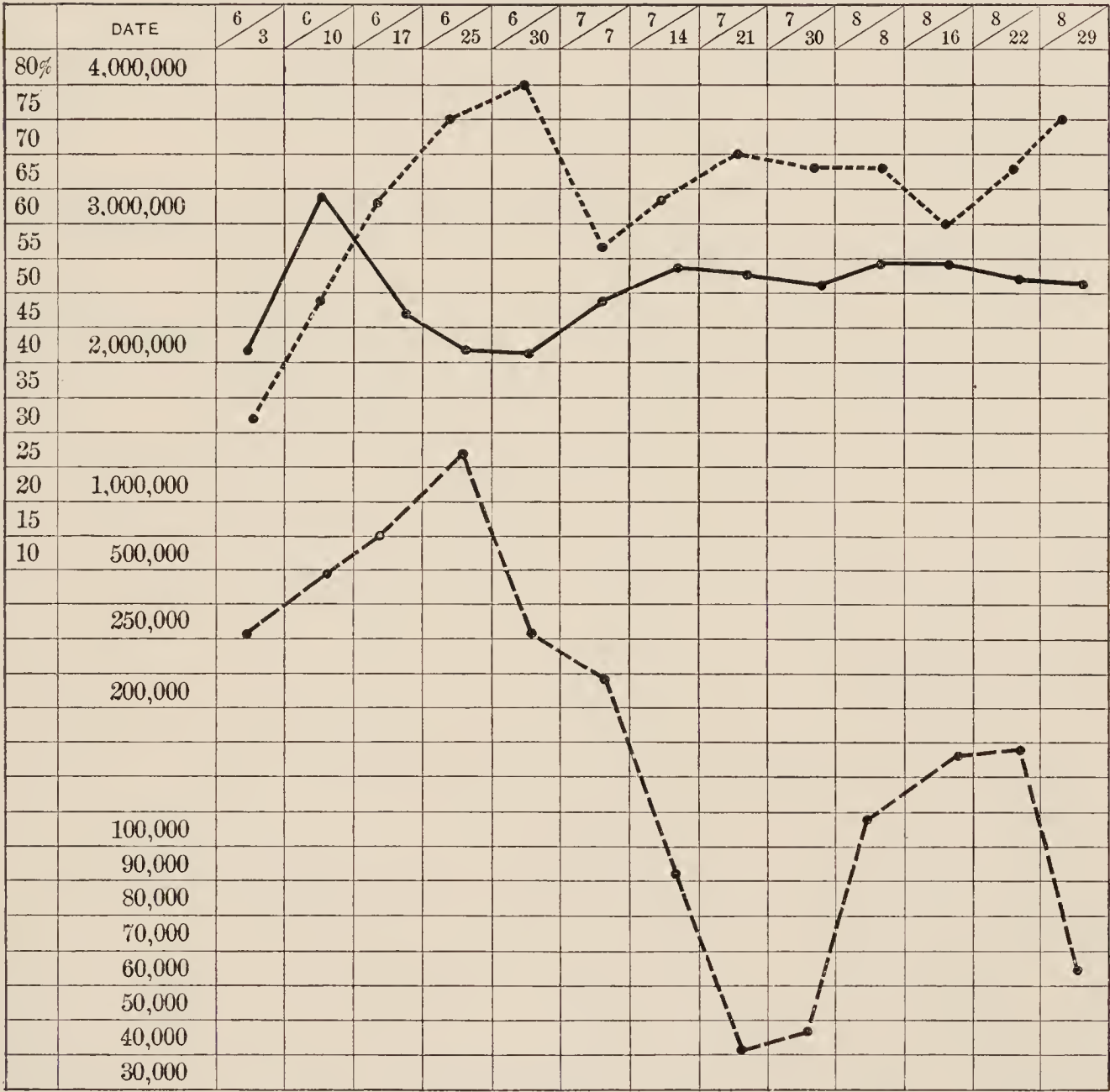
Spleno-Myelogenous Leukæmia.

1. Myelocyte. 2. Eosinophile Myelocyte. 3. Normoblastic Red Corpuscles: dividing or fragmenting nuclei.
4. Eosinophile Leucocyte. 5. Large Mono-nuclear Lymphocyte. 6. Small Lymphocyte.
7. Polymorphonuclear Leucocyte or Neutrophile. 8. Megaloblast.

nose, gums, bowels, stomach, lungs, or uterus further exhaust the patient ; digestion is poor and diarrhœa is common. Headache and tinnitus are frequent symptoms, occasionally delirium and coma may occur, and deafness is not uncommon toward the close of the disease. The eyes may be the seat of leukæmic retinitis.

The *liver* is enlarged, often to a considerable degree, but without special symptoms. The same is true of the lymphatic glands and other adenoid tissue. (See Plate XVIII.—C, Fig. 1.) The marrow of the bones becomes the seat of disease in some cases, but it does not usually give rise to symptoms during life ; certain bones, however, may be tender.¹

FIG. 333.



Leukæmia. Continuous line, red cells ; small dots, hæmoglobin ; large dots, white cells.

The Blood. The most characteristic and important changes from a diagnostic point of view occur in the blood. The blood when drawn from the finger has often a decidedly muddy hue and may interfere with the hæmoglobin estimation, an appearance supposed at one time by Bennett to be due to admixture of pus. It coagulates slowly, is of lower specific gravity than normal, and its alkalinity is diminished. When placed under the microscope, it is at once seen that the number of white

¹See "A Case of Leucocythæmia." Musser and Sailer, Amer. Journ. of the Med. Sciences, 1896.

cells is greatly increased, and that a large percentage of these increased cells is made up of mononuclear, finely granular cells, the myelocytes, or marrow-cells. These cells are not amœboid, a distinctly noticeable feature. The granules are fine, and neutrophilic, the nucleus is oval. As many as 1,000,000 white cells may be counted in a cubic millimetre of blood, the myelocytes constituting 30–50 per cent. of the total number. If a drop of blood is mixed with distilled water containing a small quantity of gentian-violet, the white cells are stained a decided blue and can be picked out with the greatest ease. Instead of there being 1 white cell to 300 or 500 red, the ratio falls as low as 1 : 5 or 1 : 3, or even lower. Even in periods of comparative well-being, when the total leucocyte count may be nearly or quite normal, a high percentage of myelocytes still remains and distinguishes leukæmia from other conditions in which the white cells are increased. In ordinary leucocytosis myelocytes are absent, and an increased percentage of the polymorphonuclear leucocytes is regularly seen.

Not only do the white cells greatly increase in number, but they vary considerably in size and react differently to staining fluids. Ehrlich has described five varieties of leucocytes.

The variations in the leucocytes in this disease are: (1) the small mononuclear and polymorphonuclear elements are relatively diminished; (2) the great difference in size of the multinuclear elements; (3) the presence of the myelocytic elements; (4) the presence of a normal or but slightly increased proportion of eosinophiles in so extensive an increase of leucocytes¹ and the presence among these of eosinophiles with a single round or oval nucleus, the eosinophilic myelocyte, rarely seen in any other disease. (Plate XVIII.—C, Fig. 2.) (5) Large mononuclear elements with karyokinetic figures. (Müller.) (6) “Mast-cells.” Satisfactory study of these can be obtained only by cover-glass preparations.

The essential points in the diagnosis of myeloid leucocythæmia are: 1. A marked excess of leucocytes in the blood, with a high percentage of myelocytes, the ratio of white to red falling below 1 : 50 or 1 : 20. The individual leucocytes vary in size and characteristics, as already described. 2. Enlargement of the spleen. 3. The occurrence of hemorrhages and dropsies unexplainable by disease of the heart, kidneys, or other organs. 4. The symptoms of a high grade of anæmia, as dyspnœa. 5. Leukæmic retinitis. 6. Anæmic fever. 7. The presence of eosinophilic myelocytes, “mast-cells,” and nucleated red blood-cells. 8. Specific gravity below 1040. 9. Excess of uric acid in the urine. 10. Charcot-Leyden crystals in the blood.

The *lymphatic form* of the disease is not so common. It is characterized by enlargement of some group of the lymphatic glands and by the great increase in the proportion of the lymphocytes, which may be of the small or large variety or of intermediate size and average about 90 per cent. of the leucocytes present. The total increase in the colorless elements is not so excessive; 300,000 per c.mm. is an extreme. Eosinophiles and nucleated red cells are rare. The myelocyte of Ehrlich is not present. Acute leukæmias are of the lymphoid type and frequently

¹ W. S. Thayer, loc. cit.

show the greatest increase in the large mononuclear elements (lymphocytes); the main features are high fever, a duration of symptoms of only a few weeks, and a rapidly progressing anæmia occasionally with nucleated red cells.

TABLE SHOWING COMPARATIVE DIFFERENCES OF BLOOD IN VARIOUS AFFECTIONS.

Leukæmia.	Normal.	Chlorosis.	Pernicious Anæmia.	Secondary Anæmia.
<i>Red cells.</i>	4,500,000–5,000,000.	Average, 3,500,000; rarely under 2,000,000. Poikilocytosis usually only in severe cases; few microcytes and megalocytes.	Average, 1,000,000. Marked poikilocytosis; microcytes and megalocytes numerous.	Average, 1,500,000 to 2,500,000. Poikilocytosis present; microcytes and megalocytes.
Normoblasts.	. . .	Occasional.	Fairly numerous.	Numerous.
Megaloblasts.	. . .	Rare.	Usually present in severe conditions.	Sometimes present.
Hæmoglobin.	90–95 per cent.	As low as 30–40 per cent.	Relatively higher than red cells.	Relatively lower than red cells.
<i>White cells.</i>	4000–10,000.	Normal or diminished. 2000–8000.	Normal or diminished.	Usually increased.
Small lymphocytes.	20–30 per cent.	Slightly increased.	Increased.	Diminished.
Large lymphocytes.	5–6 per cent.	Increased.	Increased.	Diminished.
Polymorphonuclear.	60–75 per cent.	Slightly diminished.	Diminished.	Increased.
Eosinophiles.	2–4 per cent.	Normal.	Normal.	Normal.
Myelocytes.	. . .	Rare.	Present in severe cases.	Present in severe cases.

	Pathological Leucocytosis.	Leukæmia, Splenomyelogenous.	Leukæmia, Lymphatic.
<i>Red cells.</i>	Normal or diminished.	2,000,000–3,000,000.	2,500,000 and less.
Normoblasts.		Numerous.	Comparatively rare.
Megaloblasts.		Present at times.	Rare.
Hæmoglobin.	Normal or diminished.	Relatively diminished.	Diminished.
<i>White cells.</i>	Usually not over 50,000.	Average, 200,000–400,000; 1 : 10, 1 : 5, or even 1 : 1 with red cells.	Average, 100,000 or less.
Small lymphocytes.	Diminished.	Diminished.	Increased; 90 per cent. or more.
Large lymphocytes.	Diminished. Together about 15 per cent.	Diminished. Together about 10 per cent.	Increased. Either small or large may predominate.
Polymorphonuclear.	Average, 85–90 per cent.	Diminished 30–40 per cent.	Diminished; average, 3 per cent.
Eosinophiles.	Normal or diminished.	Increased; 4–10 per cent.	Very scarce.
Myelocyte.	Occurs at times.	Increased; 35–50 per cent.	Rare; 0.3 per cent.

In Hodgkin’s disease, lymphadenoma, there are marked and early glandular enlargement and progressive anæmia, the white cells are generally normal in number and in relative percentages toward the end, or if inflammatory changes supervene they may rise from 60,000 to 70,000 per c.mm. This is, however, about the limit of their increase and the relative increase of the polymorphonuclear cells is often a marked symptom. Syphilitic and tuberculous adenitis must be excluded; the spleen, though enlarged in these conditions, rarely reaches the size seen in leukæmias.

“Splenic anæmia,” “splenic pseudoleukæmia,” “splenomegalic primitive,” or, as it has been wrongly called, the “splenic type of Hodgkin’s

disease," resembles the leukæmias in general symptoms and in the presence of splenic enlargement, which is often as large as that seen in myelogenous leukæmia. Removal of the spleen has improved or even cured some cases; hence "anæmia with enlarged spleen" is a more acceptable term. The anæmia is marked and progressive, the white cells are diminished (leucopenia) and show no special variations; the reduction of hæmoglobin is excessive and the corpuscular color-index is below normal. Poikilocytosis and nucleated red cells are not present. Banti's disease, if such exists, presents the same characteristics with the addition of cirrhosis of the liver developing after the enlargement of the spleen.

Anæmia infantum pseudoleukæmia of von Jaksch, is disputed as a clinical entity; an intense anæmia, leucocytosis, large spleen and liver, a blood condition with certain characteristics of both leukæmia and pernicious anæmia or even of secondary anæmia are to be seen. It is remembered that the infantile hæmatopoietic system responds readily to any influence with a result far more striking and varied than in the adult. Many of von Jaksch's cases may be leukæmias, pernicious anæmias, or anæmias secondary to rickets or syphilis; some doubtless, according to the condition of their blood, belong to some intermediary state; poikilocytosis, presence of normoblasts and megaloblasts, lymphocytosis, and a moderate percentage of myelocytes, may coexist.

Hodgkin's Disease.

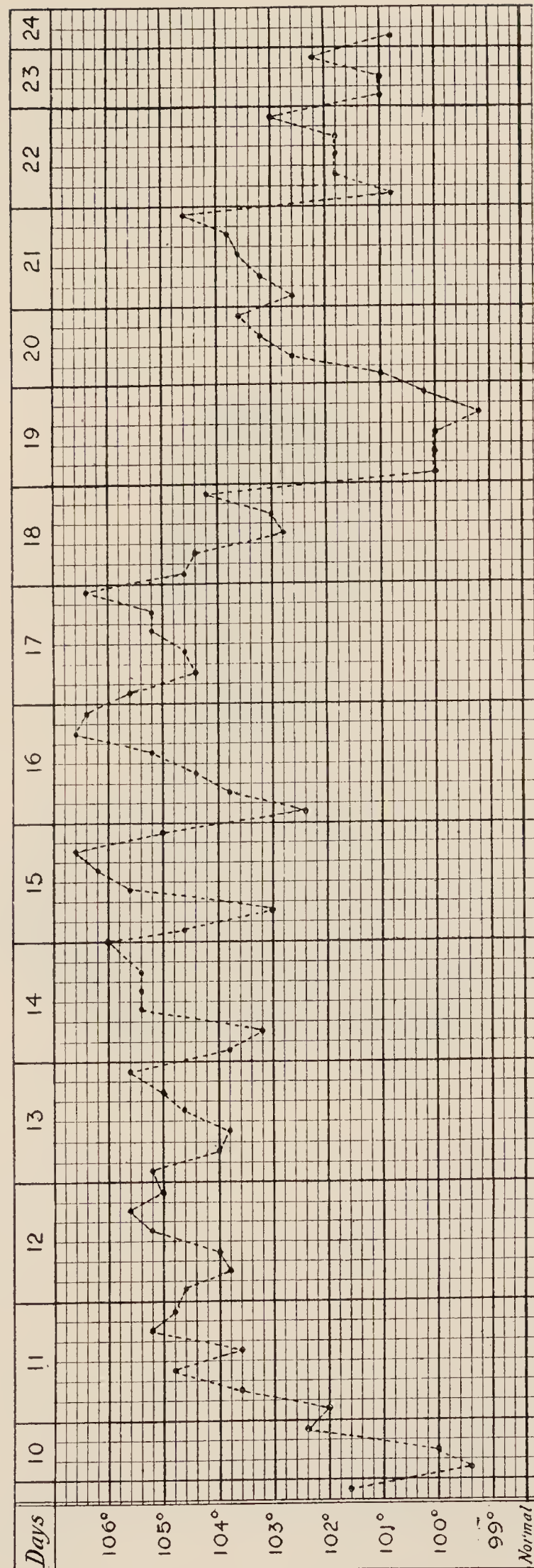
Hodgkin's disease, an extremely rare affection (pseudoleukæmia, lymphadenoma, or lymphatic anæmia), is characterized by enlargement of the lymphatic glands and other adenoid tissue; by progressive oligocythæmia, without, in most cases, much increase of leucocytes, and by the development of lymphatic tumors in unusual situations. The disease is most frequent in the first half of life and three-fourths of the cases occur in males.

The first symptom noted is *enlargement of the glands* of the neck; but sometimes the inguinal, less frequently the axillary glands, are first enlarged; rarely the tonsils are the first to be affected. The enlargement is painless and progressive, appearing first on one side of the neck and extending under the jaw to the opposite side. The tumors at first are distinct, and movable under the skin. The swollen glands may remain in this condition indefinitely for months or years; but eventually they begin to enlarge rapidly, lose their separate identity, and coalesce into large masses. Other glands in remote parts, as the axilla and groin, retroperitoneum, and arm, are affected. They may be soft and fluctuating, or very dense and hard, but heat, tenderness, suppuration, and other evidences of inflammation are absent.

The spleen becomes greatly enlarged, but rarely attains the dimensions common in leucocythæmia. Other adenoid tissue in the intestine, tonsil, and posterior nares, and even the thymus, may enlarge and give rise to pressure symptoms.

Fever is a very constant symptom, but the type is not constant. It may be intermittent, remittent, relapsing, or continuous. A curious form

FIG. 334.

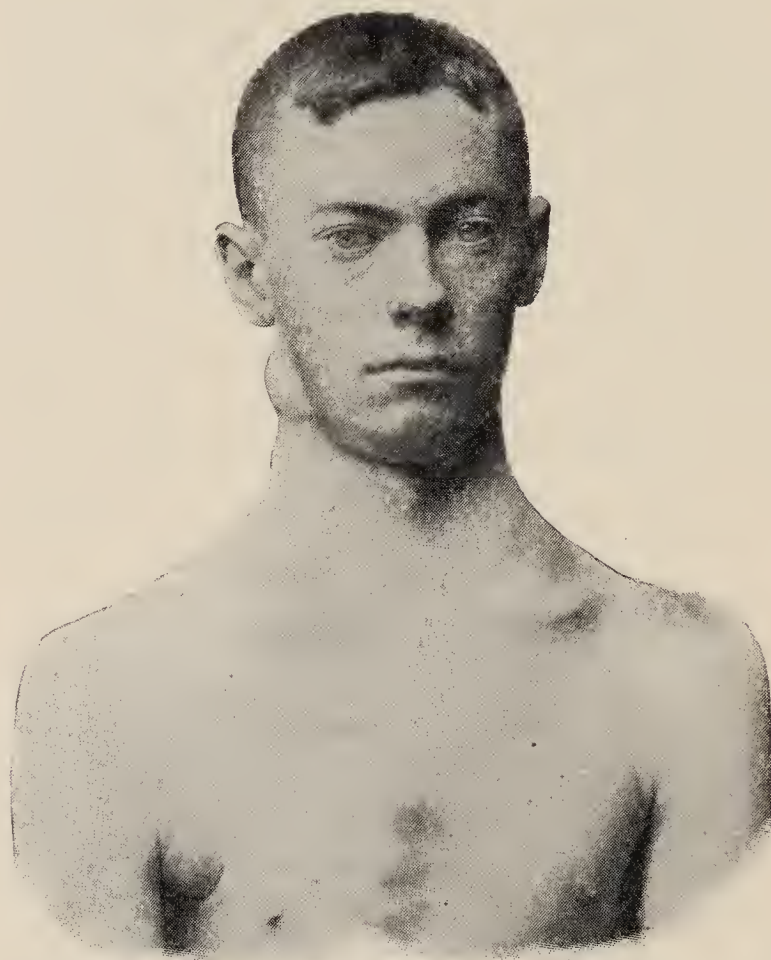


A characteristic febrile period in the recurrent fever of Hodgkin's disease.

is that known as *Ebstein's disease*, "Rückfall," or the relapsing type in Hodgkin's disease. The accompanying chart illustrates the course of the fever during one of the paroxysms. In the interval, which may last ten days or more, the temperature is normal. The paroxysms last from

three to ten days. The onset of the disease may be marked by fever and constitutional symptoms, and the glandular enlargement appears later. On the other hand, in 3 cases reported by J. Dreschfeld,¹ all the patients enjoyed good health and were able to continue their work until a few weeks before death. In all, the symptoms appeared suddenly, and consisted of pain, weakness, pallor, loss of appetite, and pyrexia. Coincident with the rapid and extensive enlargement of the glands, anæmia may arise accompanied by the usual symptoms. Cough is often associated with anæmic dyspnœa, and in women menstruation may cease.

FIG. 335.



Hodgkin's disease. Glands in right axilla and neck much enlarged. (Original.)

Along with the general symptoms there are numerous local ones, due to pressure or to impairment of function—cerebral anæmia from pressure on the carotids; cerebral congestion from pressure on the veins of the neck; disturbance of the heart from pressure on the pneumogastric; deafness; difficulty in deglutition and mastication; and pleural, peritoneal, and pericardial effusions.

The most frequent *complications* are nephritis, fatty degeneration of the heart, pleurisy, and, less frequently, pneumonia and pericarditis.

The *duration* of the disease is from six to eighteen months. Two-thirds of fifty fatal cases referred to by Gowers² ended in less than two years. It is difficult to determine accurately the beginning of the disease; sometimes a long period of latency follows the early glandular swelling; sometimes a general anæmia precedes any noticeable swelling of the glands; and sometimes the disease runs an acute course, ending fatally in two or three months.

¹ British Medical Journal, April 30, 1892.

² Reynolds' System of Medicine, Philadelphia, 1880, vol. iii., p. 549.

Death results most frequently from exhaustion ; but pressure upon the trachea producing asphyxia is not uncommon, and death has occurred from starvation, the result of occlusion of the œsophagus by pressure. The complications already mentioned are the immediate causes of death in other cases.

Diagnosis. The diagnosis is not difficult if a blood examination is made. By this means leucocythæmia is excluded. It may be distinguished from tuberculosis in the early stages when local by the site of the enlargement. In the former the submaxillary glands are involved ; in the latter the glands in the anterior and posterior cervical triangles. The *tuberculin* test is required, as insisted upon by Otis, to establish tuberculous adenitis.

Purpura.

Primary purpura occurs without any known cause. It has been divided, for convenience, into *simple* and *hemorrhagic* purpura, though the two differ only in intensity.

Secondary purpura occurs in connection with a variety of febrile and constitutional diseases : (1) scurvy, (2) hæmophilia, (3) Hodgkin's disease, (4) splenic leucocythæmia, (5) pernicious anæmia, (6) chronic lesions of the kidney and liver, with or without jaundice, (7) ulcerative endocarditis, (8) malignant sarcomata, and (9) infectious diseases.

In *simple purpura* the hemorrhages are limited to the skin. They consist of : (1) bright-red spots, varying in size from that of a pin-head to that of a silver three-cent piece. These spots are under the skin and are unaffected by pressure ; they fade gradually from red to yellow and disappear. (2) Larger spots or streaks called vibices. (3) Ecchymoses. The disease is said to be most common about the age of puberty. It may come on in the midst of apparent health, or it may follow an illness, as typhoid fever. Purpura occurs especially upon the legs, the standing position seeming to favor its development. The eruption comes on in successive crops. Sometimes large blebs, filled with thin blood, form under the skin, and gangrene occurs at times.

In the *hemorrhagic form*¹ hemorrhages occur from the nose, stomach, bowels, vagina, and bronchi, or into the kidney or some other viscus. Cutaneous and submucous hemorrhages also occur. The onset of these cases is sudden, though there may be a day or two of depression, lassitude, headache, and nausea. The first symptom noticed is generally, *fever*, which is apt to be moderate, then an *eruption* is detected, and for a day or two the patient may seem to be only slightly ailing. Copious epistaxis may now occur, or a hæmatemesis or hæmaturia, or all of these and other hemorrhages may occur the same day. The temperature may be only moderately raised, or it may reach 104° to 105° F., or even a higher point. The pulse at first is frequent (120 to 140), but of good volume and tension. Subsequently, in unfavorable cases, it becomes thready and very frequent. Respiration is not affected, and the mind is

¹ See "Grave Forms of Purpura Hæmorrhagica." Musser, Trans. Association of American Physicians, vol. vi.

clear; the face is pale and anxious. Hemorrhage may also occur into the choroid and brain-substance, with blindness and paralysis as sequels. It may also occur into the uvula or tonsil.

The *subjective symptoms* are pains in the loins, limbs, epigastrium, or chest. Often these pains announce a fresh hemorrhage, as into the kidney, or a fresh crop of purpuric spots. The degree of anæmia depends upon the copiousness of the hemorrhage and the length of time the disease lasts. Sometimes the hemorrhages cause great exhaustion, with a tendency to collapse. In some instances gastro-intestinal *crises* of severe pain and hemorrhage occur, with occasional *arthritis* and acute *nephritis*. The *urine* in the case of hemorrhage into the kidney, of course, contains blood; sometimes casts are also found.

Peliosis Rheumatica. Another variety of purpura is known as peliosis rheumatica, the peculiar features of which are tender and swollen joints, œdema of the subcutaneous cellular tissue, and purpura associated with urticarial wheals and intense itching (purpura urticans). The subcutaneous hemorrhages consist of petechiæ, vibices, and ecchymoses. There may be such large hemorrhages into the penis, scrotum, and uvula as to result in gangrene and slow separation of the dead tissue by ulceration. Epistaxis may occur, but copious hemorrhages from the stomach, the bowel, or into the kidney or other organs are rare. Endocarditis and pericarditis occur as complications in some cases. The duration is apt to be long, convalescence being delayed by repeated outbreaks of purpura with multiple arthritic symptoms and œdema.

Diagnosis. Primary purpura is distinguished from *scurvy* by the absence of antecedent debility and anæmia, of spongy gums, of brawny induration in the limbs, and by the fact that the hemorrhages do not usually occur around a hair-follicle. In scurvy there is a history of deprivation of vegetable food, whereas purpura may occur in the midst of robust health. As a rule, the cutaneous hemorrhages are larger in scurvy than in purpura.

Purpura is distinguished from *acute infectious diseases*, particularly typhus, cerebrospinal fever, and smallpox, by the absence of severe constitutional symptoms which characterize the graver forms of these diseases—in which alone a purpuric eruption is likely to be severe enough to cause doubt. Hemorrhages from mucous surfaces are rare in the latter.

Hæmophilia is distinguished by the history the patient gives of being a bleeder by heredity, and the fact that the bleeding has been started by some injury, wound, or operation.

It is distinguished from the hemorrhages of *leukæmia* by the absence of enlarged spleen and liver, and by the fact that there is no excess of leucocytes in the blood.

Malignant sarcoma causing hemorrhages is recognized by the previous history of anæmia and cachexia, and by the detection of primary or secondary growths.

It must not be confounded with *Raynaud's disease*, a vasomotor affection characterized by local syncope, local asphyxia, and gangrene.

Hæmophilia.

The diagnostic significance of subcutaneous hemorrhage is clearer when associated with profuse hemorrhages in other portions of the body, and when there is also a history of the occurrence of such hemorrhages in the family. Hæmophilia is a constitutional affection characterized by bleeding, which is spontaneous or occurs upon slight injury. It is nearly always hereditary, but may arise *de novo*. Males are very much more liable to it than females, the ratio being about 11 to 1. This curious disposition to bleeding may be transmitted for generations, and almost always to the males through the female members of the family—that is to say, the daughter of a bleeder is not usually affected, but she transmits the tendency to her sons, who become bleeders; so, too, the granddaughters are not bleeders, but they in turn transmit the disposition to their male offspring. It generally shows itself early in life, usually before the end of the second year, and almost invariably before puberty is passed.

The affection usually first declares itself by the occurrence of a *hemorrhage*, either spontaneous or the result of slight injury, the bleeding being far more profuse than would be natural, and in some cases absolutely uncontrollable. Legg¹ has divided hæmophilia into three degrees, according to the severity of the symptoms. The first is characterized by external and internal bleedings of every kind, and by joint-affections; the second, by spontaneous hemorrhages from mucous membranes, but no traumatic bleeding or ecchymoses, and no joint-affections; the third, by a tendency simply to ecchymoses. The first form is seen most frequently in men; the second most frequently in women; and the third in either sex.

The most frequent *seat of hemorrhage* is the nose, and next the gastrointestinal tract. The bleeding is from the capillaries; it may prove fatal in a few hours, or last for days or weeks, with final recovery. Intense anæmia follows the prolonged hemorrhage, but the blood is replaced with remarkable rapidity. All operations, even the most trivial, are extremely dangerous in bleeders. Circumcision, extraction of teeth, and leeching are credited with the most deaths by Grandidier.

Joint-symptoms are very common. The knees, elbows, ankles, and shoulders are the ones most frequently involved. The attack may be marked by pain, redness, swelling, inflammation, and fever; or fever may be absent; or pain alone may be complained of. The attacks are liable to recur, especially in cold, damp weather, and may result in stiffened, deformed joints.

Diagnosis. The diagnosis is easy when the history of a hereditary tendency to bleed can be obtained. Osler² properly remarks that slight joint-trouble and petechiæ are as much a manifestation of the disease as the more severe hemorrhages. In cases in which no history can be secured, the diagnosis is made by noting a persistent liability to hemorrhage, without adequate cause, and associated with joint-affections.

¹ Hæmophilia. London, 1892.

² Quoted by Osler, Pepper's System of Medicine, 1885, vol. iii., p. 937.

Osler gives the following excellent summary of the affections with which hæmophilia may be confounded :

1. The umbilical hemorrhages of infants, due to jaundice or to syphilis, hæmorrhagica neonatorum, etc.

2. Purpura simplex, often seen in debilitated, rarely in healthy children, usually confined to the legs, and in some cases associated with rheumatic pains or swellings in the knees and ankles.

3. Peliosis rheumatica.

4. Purpura hæmorrhagica, morbus maculosus Werlhofii, a grave disease, characterized by extensive cutaneous ecchymoses, mucous hemorrhages, but not dependent on any local disease or on any known specific poison.

5. Infective purpura due to the action of some specific poison—smallpox, measles, scarlet fever, cerebrospinal fever, etc. The hemorrhages may be cutaneous and trivial, or may be in the most aggravated form of interstitial and mucous bleedings, as seen, for example, in black smallpox.

6. Toxic purpura, as in snake-bites and many poisons, such as phosphorus.

7. Simple hemorrhagic diathesis, under which may be included those cases in which, without any hereditary disposition or previous hemorrhagic history, there is a tendency to uncontrollable hemorrhage from a slight wound.

8. Hæmatidrosis, bloody sweat, which occurs usually in hysterical or epileptic females, and is in rare instances accompanied by mucous hemorrhages.

Scurvy.

Scorbutus, or scurvy, is a constitutional condition brought about by a long-continued diet deficient in fresh vegetables, or in infants by artificial foods. It is characterized by pallor, great physical weakness and mental sluggishness, dyspnœa, subcutaneous and submucous hemorrhages, a swollen, spongy condition of the gums, and a brawny induration, especially of the calves and hams.

The *joints* are swollen, painful, and tender in about one-third of all cases of scurvy. When to these joint-symptoms the spongy gums, the hemorrhages, the anæmia, and cachexia are added, scurvy may be suspected. The onset of the disease is gradual, and is marked by a peculiar dirty yellow or greenish *pallor* of the face, associated soon with an apathetic expression of the face, physical *weakness*, and decided lack of customary energy. The appearance is so characteristic that patients are said to detect it readily in others, though unaware of it themselves.

The *gums* swell almost always, become spongy, and bleed upon slight irritation. They are dark cherry-red in color and look not unlike a split cherry. Sometimes they swell so as almost to hide the teeth completely and even to protrude the lips. The breath has a heavy, sickening odor, and the teeth sometimes drop out of their sockets. In some cases the eye and its surroundings are the only parts exhibiting signs of scurvy at this time. "The integument around one or both orbits is puffed up into a bruise-colored swelling. The conjunctivæ covering the sclerotic is

tumid and of a brilliant red color throughout, and about an eighth of an inch in thickness or in elevation above the cornea, the latter being at the bottom of a circular trench or well.”¹ The condition is not inflammatory. These cases often terminate fatally.

Sleep and digestion are good, but rheumatoid pains may be complained of. Other prominent subjective symptoms are fatigue on slight exertion, dyspnoea, faintness, and despondency. In the course of a week or two petechiæ appear upon the lower extremities, especially around a hair as the centre. (See page 211.) Depending upon the severity of the case there are also bullæ, vibices, and ecchymoses. Brawny induration, due to deep effusion of blood, occurs, especially in the calves and hams, producing considerable pain on flexure of the knees.

There is no fever apart from complications. The pulse is frequent, weak, and small, and the first sound of the heart and the impulse may be very faint.

In addition to the cutaneous and gingival bleeding, hemorrhages occur from the nose and other mucous surfaces, and effusions take place into the lungs, intestines, pericardium, and pleura, associated with inflammatory products. There may be no physical signs of the lungs to account for the dyspnoea; or some dulness and bronchial breathing, or a few râles, may be detected.

A very peculiar symptom, and sometimes the earliest, is *hemeralopia*, or night-blindness; the patient can see during the day but not by moonlight, and except with artificial light is totally blind at night.

So-called *scurvy-rickets* is more or less common in infants fed on artificial food exclusively or on sterilized milk. It is therefore limited to the first four or five years. The symptoms of scurvy are added to those of rhachitis. In 15 cases I have seen, the most pronounced features were those of weakness, anæmia, polyuria, restlessness, the scorbutic gums, local periostitis, particularly of the tibia, sometimes periarticular inflammation, and always a general tenderness of the body, as in rhachitis. In all of them the presence of a dietetic cause confirms the diagnosis.

DISEASES OF THE THYROID GLAND.

The thyroid gland may be enlarged from simple hypertrophy or from fibrocystic disease. The characteristics of these affections are considered in Chapter XX., Part I. Enlargement may also be due to abscess and morbid growths, which are considered at the end of that chapter.

Enlargement of the Thyroid.

Enlargement of the thyroid can be detected without much difficulty. It may be limited to one lobe, or both lobes may be affected. It may vary in size from a small localized swelling to large masses which fill the median and lateral sides of the neck, pressing upon the trachea and extending into the thorax. On palpation the swelling may be

¹ Buzzard, Reynolds' System of Medicine, 1880, vol. i., p. 451.

soft or hard. In the fibrous forms the swelling is not very large and is very much indurated. In the cystic forms of the thyroid enlargement, fluctuation may often be detected ; it may be localized to a small area of the lobe, or may be detected over the entire affected lobe. In some cases, on palpation, a purring or thrill is transmitted to the fingers. The thrill is synchronous with the heart's action and due to increased vascularity of the gland. Auscultation under these circumstances reveals a systolic murmur.

Causes. Enlargement of the thyroid gland may be due to simple hypertrophy, to fibrocystic enlargement, or to enlargement in which the vascularity is more prominent, as in exophthalmic goitre. 1. In simple *hypertrophy* the enlargement is often intermittent, increasing in size at each menstrual period, or coming on in pregnancy to disappear after labor. It may then disappear entirely or return at the menopause. 2. The *fibrocystic* enlargement which occurs in countries in endemic form is persistent. 3. The enlargement of exophthalmia generally continues throughout the course of the disease. (See below.)

Exophthalmic Goitre.

Exophthalmic goitre, Graves' or Basedow's disease, is far more frequent in women than in men. It may develop at any age, but is most common in early adult life. A neurotic heredity, exhausting disease, general debility, and anæmia are predisposing causes, while sudden fright or shock is the most common exciting cause. Graves' disease begins slowly.

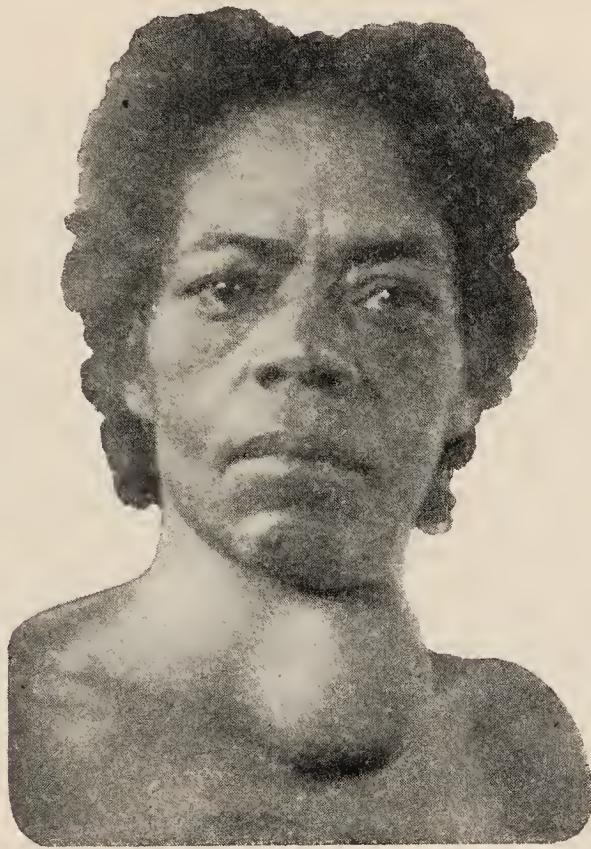
Of the three classic symptoms, *rapidity of the heart's action*, with palpitation, *enlargement of the thyroid*, and *prominence of the eyes* (exophthalmos), the first is the essential symptom. It is also usually the earliest. Either enlargement of the thyroid or exophthalmos may be absent for months or years, and in some instances throughout the disease.

Tachycardia. Attacks of palpitation may recur at intervals for a long time before their true nature is suspected. In these attacks the behavior of the heart is much like that which occurs under the influence of fright or great excitement. The frequency may not be over 100 or 120 in the early attacks, the rate being normal in the intervals. In the later and severe attacks, however, the pulse beats 160 or 180 or even 200 per minute. It is small and regular. The heart beats with increased force ; the sounds are loud, sharp, and clear, occasionally being heard several feet from the patient. In time the heart becomes hypertrophied and dilated, and there is often a loud, basic, systolic murmur. The larger arteries and even sometimes the smaller ones show the vascular disturbance by increased pulsation, sometimes with thrill.

The Thyroid Gland. The thyroid is usually the next to become affected. It enlarges slowly from vascular dilatation, the swelling at first subsiding in the intervals between attacks, but subsequently persisting. The right lobe may be larger than the left. The enlargement is painless, soft, and compressible. It may pulsate with or without thrill, and over it can be heard hæmic murmurs.

The Eyes. Prominence of the eyes is the most conspicuous feature of well-marked cases. Like enlargement of the thyroid, it varies in degree, and rarely is wholly absent. The protrusion allows the white sclerotic to show above and below the cornea, giving the eyes an unnatural, startled, staring appearance. The protrusion may be so great that the eyelids can not close; more commonly they close, but when the eyeball is simply directed downward the upper eyelids do not follow but remain spasmodically elevated or lag behind the movement of the eyeball. (Von Graefe's sign.) The eyeball may become inflamed and even slough from undue exposure. In rare instances one eyeball alone is

FIG. 336.



Exophthalmic goitre. (Original.)

affected, and in these cases the lobe of the thyroid of the opposite side is enlarged. Stelwag's sign (widening of the palpebral fissures) is the third ocular sign of significance in exophthalmic goitre. Finally, Möbius calls attention to the frequency of insufficiency of the internal recti muscles.

In addition to these characteristic symptoms *loss of flesh* and *strength*, moderate pyrexia of irregular type, impaired appetite, diarrhoea, and despondency are observed. The *diarrhoea* is of the nervous type—increased peristalsis without local catarrh. The menstruation is apt to be disturbed. Tinnitus aurium, headache, and vertigo are not uncommon, and sometimes there is profuse *sweating*. A restless, nervous excitement (Charcot) is very common. *Muscular tremor* (Marie), occurring on voluntary movement, is frequently observed, and with diarrhoea is almost as common as the three primary symptoms. *Œdema* of the feet is often seen if there is coexisting mitral disease. Transitory vasomotor œdema of the eyelids, the face, hands, and the supraclavicular and infraclavicular regions occurs. It is usually circumscribed, and may not pit on pressure. Transient erythemas are not uncommon.

Prognosis. Graves' disease, as a rule, runs a chronic course, lasting for years. A few cases that have run an acute course of a few weeks, some ending in recovery and some in death, have, however, been reported. Moreover, there may be recurring attacks with apparent recovery in the intervals. Death results from gradual weakening of the heart and its direct and indirect effects. It may be hastened also by uncontrollable diarrhœa, acute mania, and epilepsy. The disease may also be complicated with hemorrhages, and these may be the immediate cause of death.

Enlargement of the thyroid gland from the above-mentioned causes must be distinguished from enlargement due to *abscess, cancer, sarcoma, or adenoma*. Abscess usually follows infectious diseases; in the writer's case it followed typhoid fever. With carcinoma and sarcoma there are anæmia, gradual loss of flesh, and the usual clinical phenomena of these processes. It must also be distinguished from other tumors in this region. It particularly must not be confounded with enlargement on the right side due to an innominate aneurism. (See Aneurism.)

Myxœdema.

Myxœdema is a disease of mature life and occurs most frequently in women. The swelling is general and involves the face. The arms are more markedly swollen, however, than the fingers; the legs more than the feet. Usually the swelling of the legs and arms is irregular. In some cases *supraclavicular paddings* are marked. These paddings must not be confounded with the pseudolipomata described by Verneuil as occurring in these situations. The swelling is due to infiltration of the connective tissue with mucin, and arises from some affection of the thyroid gland. The *gland is absent* functionally or actually. The hard, indurating, non-pitting swelling is associated with striking change in the appearance of the face, particularly the nose and forehead. The nose becomes thickened, the forehead more prominent and overhanging. The outline of the face is rounded, and the term "full-moon" is applied to it. The *skin* is thickened, dry, and rough, somewhat translucent, pale or yellow in color, and of a doughy consistence, but with a moderate degree of elasticity. The perspiration is diminished. The hands change in shape, become square or spade-shaped, and the fingers clubbed. The nails become brittle and distorted; the hair dry, harsh, and may fall out.

With these remarkable changes in the exterior, marked nervous and mental symptoms arise. *Speech* is thick and hesitating, the memory feeble. The *intellect* is dull and irresponsive; the temper irritable. *Sensibility* is impaired, particularly the pain sense. Patients have been burned without their knowledge, as happened in one of the writer's cases. Abnormal sensations of heat and chilliness are complained of, as well as other paræsthesias. The patient is anæmic, the temperature is subnormal, the heart's action weak, the respiration sluggish. Breathlessness on slight exertion is pronounced, and exertion itself is very difficult; while there is a greater sense of fatigue than the exertion and the condition of the organs would warrant. The muscularity is enfeebled. There are impairment of appetite, indigestion, and flatulence.

The *urine* may become albuminous, but for a long time is not characteristic save in amount and specific gravity. The former is increased, the latter lowered.

FIG. 337.



A typical case of myxœdema. (STARR.)

As the case advances mental and physical failure becomes more pronounced, the patient becomes subject to hallucination, and extremely irritable. Stupor sets in; death may take place in coma or from uræmia. Myxœdema is a disease of mature life, and occurs most frequently in women.

The following varieties are seen: (1) spontaneous myxœdema of the adult; (2) infantile myxœdema; (3) operative myxœdema; and (4) endemic myxœdema or cretinism. In infantile myxœdema the functions of the thyroid body are suppressed during the period of the individual's development. Typical cases justify the name of myxœdematous idiocy.

DISEASES OF THE SUPRARENAL GLANDS.

Tumors of the suprarenal glands are considered in the discussion on Physical Diagnosis of Diseases within the Abdomen, Chapter XXXVI.

Addison's Disease. (See Plate II., page 209.)

The most marked form of *bronzing* is seen in Addison's disease—an affection characterized by a gradual loss of strength without much loss of

flesh ; by gastric uneasiness and occasional vomiting ; feeble circulation, and a bronze hue of the skin.

Social History. The disease occurs most frequently during the active period of life, from the age of twenty to forty years, and nearly twice as often in males as in females.

Asthenia. The disease begins insidiously with gradual and progressive loss of *strength*. It becomes evident from the patient's languor, weariness on slight exertion, and inaptitude for mental effort that he is suffering from some exhausting disease. The most characteristic symptom is the extreme prostration without obvious cause. Any exertion requires great effort and may induce *syncope*.

Gastric Symptoms. The appetite is impaired or lost, there is more or less discomfort at the epigastrium, and occasional vomiting.

Perhaps at this time a close inspection may show some discoloration of the skin, but usually this appears later. By degrees the gastric symptoms become more prominent, and vomiting may be so frequent as to shorten life materially. Finally the patient is unable to leave the bed. Dull pains in the head, back, and abdomen are not uncommon ; neuralgic pains in the limbs may be complained of ; and Osler states that there is tenderness on pressure in the lumbar region in a considerable proportion of cases.

The *pulse* is extremely small and feeble ; in the later stages it may be absent at the wrist.

Bronzing. The discoloration of the skin is the most striking symptom of the disease when it is well marked. The external surfaces are changed in hue, and delicate portions of the skin underneath the clothing are also bronzed. The discoloration is not removed by pressure. The areas are irregular in shape. The skin is soft and pliable. The pigment which causes the discoloration is deposited in the rete Malpighia. The pigmentation is never seen in the cornea or in the nails. The axilla, the flexure of joints, the median line, the areola about the nipple and other normal areas of pigment deposit are the common sites. Bronzed areas in sharply circumscribed patches are also seen in the mucous membrane of the lips and cheeks. Sometimes the whole body becomes of a walnut-juice color, a bronzing which is deeper in exposed surfaces. At times only portions of the body are discolored, in which case the dark hue shades off gradually into the normal hue of the skin. Wilks¹ states that in all the cases which he has seen the scalp, finger-nails, soles of the feet, and palms of the hands escaped pigmentation.

Nevertheless, discoloration of the skin is not an invariable symptom of the disease ; in some cases it is entirely absent. These cases, especially if associated with much vomiting, run a more acute course than the others, lasting only a few weeks. Such cases have been mistaken for *typhoid fever*. On the other hand, diseases of the suprarenal capsules not usually associated with the Addison symptom-complex, as carcinoma, are attended by pigmentation. However, in about an equal proportion of cases it is absent. At times the bronzing and other characteristic symptoms of Addison's disease are associated with tuberculosis in other organs. Con-

¹ Reynolds' System of Medicine, Philadelphia, 1880, vol. iii., p. 561.

versely, in cases of phthisis in which there is bronzing, tuberculous disease of the suprarenal capsules may be suspected, and the gravity of the prognosis increased.

Diseases of the spleen are considered in Chapter XXXVI, Physical Diagnosis of Diseases within the Abdomen.

Diseases of the lymphatic glands other than leukæmia and Hodgkin's disease, are considered in Chapter XXV., Part I.

CHAPTER V.

DISEASES OF THE NOSE AND LARYNX.

THE NOSE.

THE symptoms of disease of the nose are : (1) physiological ; (2) anatomical ; or, (3) those due to the morbid process.

Physiological Symptoms. Impairment of the sense of smell, anosmia, and symptoms of obstruction may occur. *Obstruction* causes retention of secretions and invites infection. Putrefaction and fermentation set in and give rise to offensive *odors*. In addition the patient becomes a *mouth-breather*. The appearance of the face is altered ; the voice changes, snoring is common, mastication is interfered with, and the amount of air that enters the lungs diminishes. (See the Lungs, Chapter VI., Part II.)

Symptoms due to the Anatomical Structure. The nose is an open space or a series of air-spaces lined with *mucous membrane*. The membrane is subject to affections that are common to all mucous membranes, and the subjective and objective symptoms are similar to those that arise in other organs, modified by the function and anatomical arrangement.

The abundance of bloodvessels and glands is the cause of one of the symptoms—namely, the *discharge*. Moreover the difficulty of removing the discharge from the various cavities in the nose in which it is pent up, leads to *obstruction*. Anatomical defects and growths produce the same effect. Because the air is constantly passing over the parts, discharges are prone to dry out, and hence *crusts* and *scabs* form. The *vascularity* of the structures of the nose is another source of symptoms. The bloodvessels are richly supplied with nerves, which cause them to contract or dilate on comparatively slight provocation by reflex action. Chilliness of the body, or of local areas of the body, chilling of the extremities, and other peripheral impressions, are followed by congestion of the nasal mucous membrane which may go on to inflammation. The vascularity predisposes to hemorrhage.

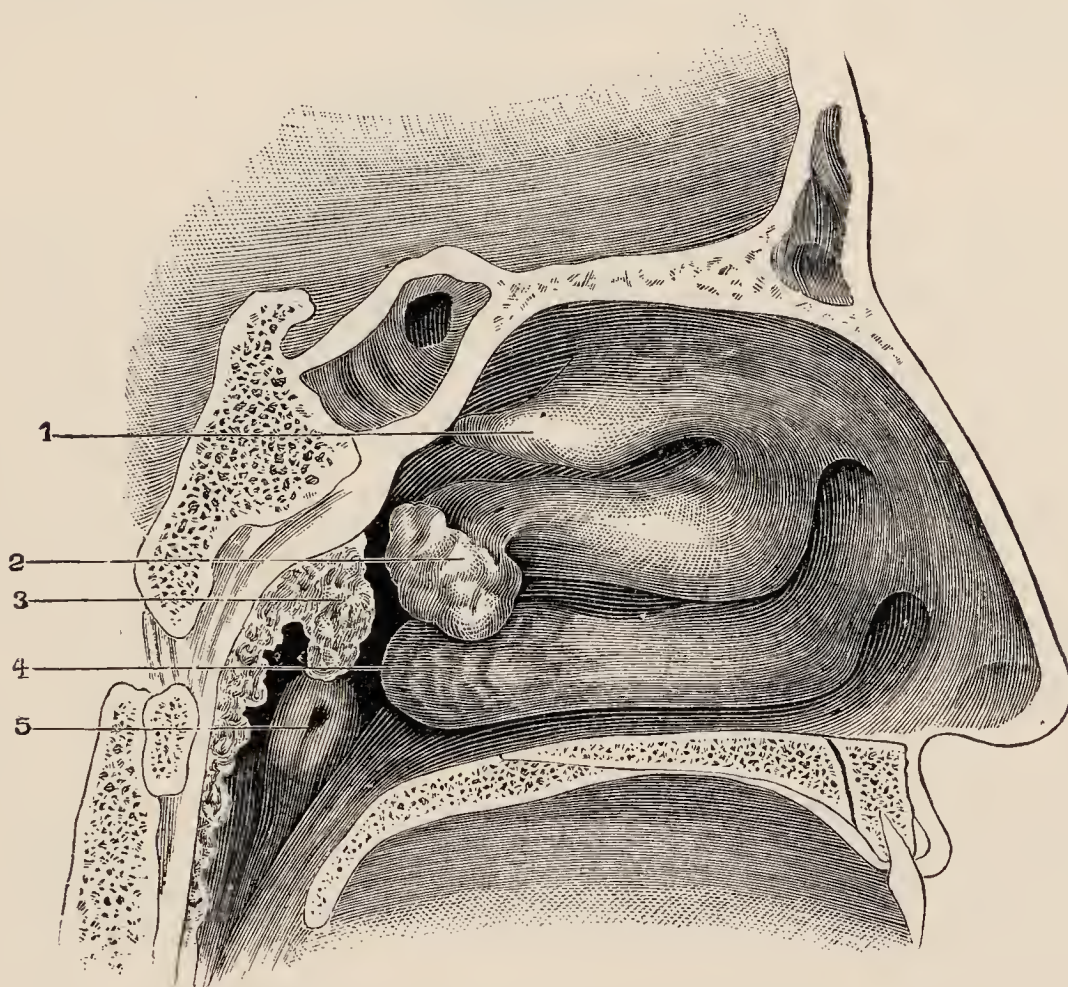
The nose is richly supplied with *sensory nerves* (in addition to the olfactory), on account of which we have the neuroses previously described. The irritation may be derived from air laden with unusual material, as fumes of a chemical nature, emanations from animals or plants, and certain substances not yet isolated, which are decidedly irritating. There is often local irritation from polyps and adenoid growths, and foreign bodies, or enlarged bone. The nerves are connected by a mechanism directly with the centres in the medulla, particularly with the pneumogastric centre. The effect of the above-mentioned peripheral nasal irritation may be felt reflexly in the area of distribution of that

nerve; hence an unpleasant odor may bring on sudden nausea or vomiting. But of more striking and frequent pathological significance is the occurrence of asthma, or sudden dyspnoea, from reflex excitation of the pulmonary division of the pneumogastric nerve.

Morbid Processes. The morbid processes are congestions; inflammations, which may be infectious, or toxic as from vapors; infections, either primary or symptomatic of some general disease; and morbid growths. Acute inflammations are significant of the exanthematous diseases, particularly measles. As Meigs has pointed out, an acute inflammation with great obstruction of the nares and an abundant, puriform discharge, is a complication or symptom of Bright's disease which may portend the onset of uræmia. Chronic inflammations may be due to syphilis or other chronic infection.

Diagnosis. The facts obtained by the *historical diagnosis* give but little information in the differential diagnosis of nasal diseases, except for the lesions which simulate one another but which have a different origin. Often only in this manner can we recognize syphilitic lesions.

FIG. 338.



Vertical section through nasal cavities. (Diagrammatic.) (SEILER.)

1. Superior turbinated bone. 2. Middle turbinated bone, with posterior hypertrophy. 3. Section of hypertrophied pharyngeal tonsil. 4. Inferior turbinated bone. 5. Orifice of Eustachian tube.

The *subjective diagnosis* demands a study of the symptoms of pain, cough, and disturbance of the sense of smell. The first two have been considered in the chapters devoted to these symptoms.

Disturbance of the Sense of Smell. (See the Nerves.) *Loss of smell*, or *anosmia*, occurs to a moderate degree in all the inflammatory and obstructive diseases of the nose. The intensity depends upon the degree of change in the mucous membrane. It may also be due to disease of the

nerves or the olfactory centre in the brain. *Parosmia* is the perception of abnormal odors, and may be a neurosis or solely a psychological difficulty and hence purely subjective, or there may be inability to distinguish an odor when presented to the nostril. All odors may appear the same, or agreeable odors may seem to the patient very disagreeable. In addition, the patient may complain of the perception of an odor in connection with the nasal disease with which he is affected. *Parosmia* is due to an involvement of the olfactory nerves.

DISEASES OF THE NOSE.

The subjective and objective symptoms previously described are due in general to inflammations, malformations, morbid growths, and foreign bodies. They are recognized by their subjective and objective signs, by rhinoscopic inspection, and by bacteriological and microscopical examinations. The inflammations may be acute or chronic, primary or secondary. When secondary, both acute and chronic inflammations may be due to infections. To the acute varieties belong the acute catarrh of measles, glanders, hay fever, or influenza; to the chronic belong syphilis and tuberculosis.

Simple Acute Rhinitis.

Acute coryza, or "cold in the head," is ushered in with a feeling of lassitude, aching in the back and limbs, feverishness, and a sense of fulness in the nostrils, with sneezing. After twenty-four hours an irritating discharge begins. During this time the malaise has increased. The pain in the forehead and cheeks has become more pronounced, and a nasal twang is given to the voice. The fever continues, the temperature reaching 101° F. in the more pronounced cases, with thirst and loss of appetite. At the height of the fever, in twenty-four or forty-eight hours, a crop of herpes very often develops on the lips. The general symptoms then subside and the local symptoms change. The discharge becomes thick and purulent, the fulness continues, but the pain is diminished. The inflammation often extends up to the tear-ducts and to the eyelids. The latter are congested and smart very much. Very frequently, also, the inflammation extends to the pharynx, causing soreness of the throat and stiffness of the neck, and the larynx even may be involved. A slight deafness may result from the inflammation extending into the Eustachian tube.

Hay Fever.

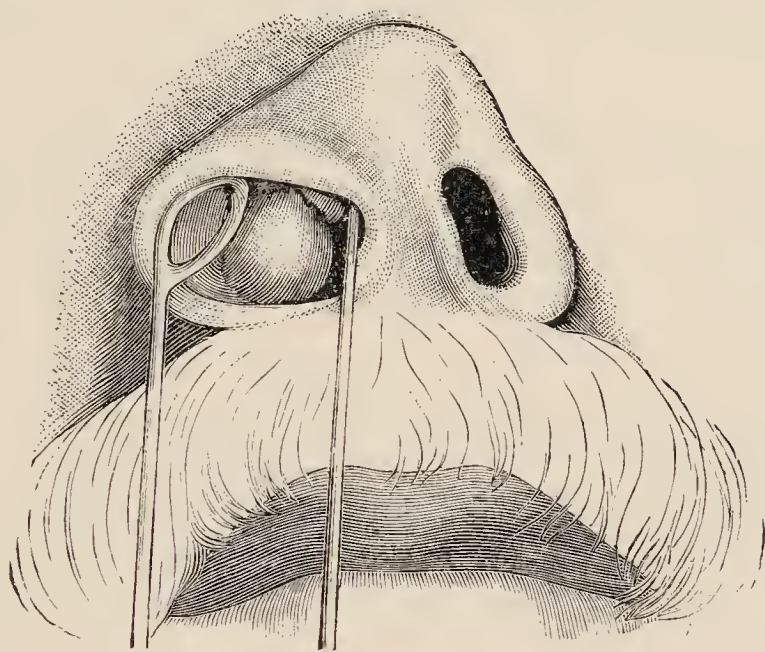
Hay fever is an acute affection ushered in by paroxysmal sneezing, itching, and smarting of the inner canthus of each eye, or of the throat or nose. After hours or days of sneezing, coryza develops. The disease continues for a varying length of time, and is more pronounced at certain seasons of the year, particularly the late fall. Coughing may be an additional symptom, and paroxysms of asthma may develop which are hard to distinguish from true bronchial asthma. The attack may be excited

by vegetable emanations, particularly the pollen of plants, but other emanations may also induce it. Certain conditions of the nasal mucous membrane predispose to the attack. Local inflammation of the nose or obstructive diseases from hypertrophies are primarily present. To the exciting cause and the local predisposing cause may also be added a neurotic factor. The disease affects families of nervous constitution, and may occur through several generations. It is more common in this country than abroad, and dwellers in cities are more subject to it than residents in the country. *Asthma* may be due to disease of the nose, but the only proof that it is of nasal origin is that it disappears after the nose has been treated for the various ailments that are supposed to cause it.

Chronic Rhinitis.

Four varieties are distinguished, to all of which the term *nasal catarrh* is applied. In one there is hypertrophy of the turbinated bones; in the second there is extension of the disease to the post-pharynx—chronic

FIG. 339.



Dilated nostril, showing anterior hypertrophy. (SEILER.)

post-nasal catarrh; in the third there is absolute dryness of the mucous membrane—*rhinitis sicca*, or dry catarrh; in the fourth there is atrophy of the mucous membrane—*atrophic rhinitis*, or *ozæna*.

Chronic Hypertrophic Rhinitis.

The affection comes on gradually after repeated acute attacks of *coryza*. The only symptoms may be slight fulness in the nose and a little hoarseness of the voice. In more advanced stages the symptoms of stenosis are marked, the patient snores and breathes through the mouth, and the voice becomes nasal. There is a constant discharge of mucus backward into the pharynx, causing hawking. The hearing is frequently impaired, as well as the taste and smell. The discharge often affects the larynx, causing an irritating cough. The hypertrophied tissue on the turbinated bones,

and the pressure of the bone on the septum may lead to reflex attacks of asthma.

Chronic Post-nasal Catarrh.

Chronic post-nasal catarrh is an extension of the rhinitis into the pharynx. It is distinguished by discomfort or pain in the soft palate and posterior nares. There are tingling and a sense of fulness at the root of the nose, with frontal headache; the patient complains of a bad

FIG. 340.



Rhinoscopic image from a case of posterior hypertrophy on the middle turbinated bone. (SEILER.)

taste in the back of the mouth, and of constant flow of thick secretion into the pharynx, causing snoring and hawking. The same perversion of the senses of taste, smell, hearing, and of the voice occurs as in acute rhinitis. Headache seems to be due to the condition of the pharynx. (See Atrophic Rhinitis.)

Dry Catarrh, or Rhinitis Sicca.

Dry catarrh, or rhinitis sicca, is also chronic in its course, and is accompanied by tingling and dryness of the nostrils. A faint, musty odor is detected, but there is no discharge or sense of obstruction. In severe cases there may be sharp pain in the nose extending to the forehead.

Syphilitic Coryza.

Syphilitic coryza is seen in infants and young children affected with hereditary syphilis. The nostrils are swollen and red at the edges, sometimes completely occluded, causing oral respiration and inability to take the breast or bottle.

Pustules, fissures, and ulcers are found in the nose and at the margin of the orifices. They are also seen in the pharynx and larynx. Hemorrhages may occur. Other evidences of hereditary syphilis are present.

Atrophic Rhinitis, or Ozæna.

The *odor* is characteristic, and is diagnostic if syphilis is excluded. A sense of dryness is complained of. Occasional obstruction arises from accumulation of crusts, otherwise the passage is unduly open. There

are constant hawking and spitting of brownish-green crusts, which are often blood-tinged. Frontal headaches may occur in paroxysms. The patient is often depressed in spirits. The bridge of the nose may fall in slightly.

The Auxiliary Cavities of the Nose.

The Antrum. The antrum is subject to abscess, cysts and polypi, tumors, and to the invasion of parasites.

Abscess. An odor somewhat like that of ozæna, a putrid taste, nausea, anorexia, pain in the cheek and at the root of the nose, often neuralgia in the frontal region, and malaise are present. A very characteristic symptom is the discharge of pus from one nostril on leaning the head forward. There is often a decayed tooth on the same side in the upper jaw.

The Sinuses. The frontal, ethmoidal, and sphenoidal sinuses are subject to inflammation, abscess, traumatism, and the irritation of foreign bodies, usually parasites.

The frontal sinuses are the only ones that exhibit external symptoms. When these cavities are inflamed, the patient complains of pain and tenderness over the frontal protuberances; if the process goes on to the formation of abscess, there may be redness and swelling and finally fluctuation. If the communication is not closed, a fetid discharge from the middle meatus takes place.

When the sphenoidal and ethmoidal sinuses are affected, there are no external symptoms unless the enlargement is so great as to affect the orbit. There is deep-seated pain. Pus is seen exuding into the superior meatus and flowing backward into the pharynx. Parasites cause intense pain and lead to abscess, caries, and necrosis. *Rhinoscopic* examination, in disease of the antrum, shows rough hypertrophic enlargement on the under surface of the middle turbinated bone and a flow of pus into the middle meatus. Sometimes a probe can be passed into the antrum from the nose. Often an exploratory puncture is necessary. *When the foramen is obstructed*, there is a dull aching pain in the upper jaw, with deformity of the orbit, face, hard palate, and nostril. Fluctuation can usually be found at some point after a time.

The *lacrymal duct* and *sac* are often the seat of inflammation by extension, causing pain, obstruction in the nose, and epiphora. On examination pus will be seen flowing forward over the inferior meatus. When the lacrymal probe is introduced, the ducts are found to be painful and obstructed, and pus exudes.

THE LARYNX.

The structural composition of the larynx does not differ from that of other parts of the respiratory passage. Mucous membrane, connective tissue, cartilages, and muscle are similar to the same tissue situated elsewhere.

The result of their anatomical association in the larynx is the establishment of the functions of that organ, the formation of the voice, and the admission of air. Now, the morbid processes that affect the larynx do

not differ from morbid processes elsewhere in which similar tissues are involved. Each tissue is liable to congestion, to inflammation, to degeneration, to new-growth formation ; the joints may become ankylosed, the muscles either paralyzed or the seat of spasm, and we have therefore all the symptoms common to morbid processes in each class of tissue. We also meet with other symptoms, which result from the anatomical position of the larynx and of its functions. The cords can not vibrate, or the muscles and articulations can not move, and *dysphonia* or *aphonia* occurs. The narrow chink of the glottis soon becomes occluded, giving rise to *dyspnœa*. Obstruction to the pathway or pain from inflammation or ulceration causes *dysphagia*. The sensitiveness of the mucous membrane provokes *cough* on the slightest provocation.

The larynx is a highly specialized organ, and is well innervated. Large central nuclei, connected by a large nerve which passes over a circuitous route and which anastomoses with other nerve-cords, preside over the function of phonation. Affections of the central nuclei, affections of the nerve-trunk or of adjacent structures exerting pressure upon the trunk, have their expression in disorder of the larynx, particularly if phonation is disturbed. In other words, the phenomena of laryngeal disease may be symptomatic of affections of the brain or of the nerve-trunk, as well as of the larynx. (See Nervous Diseases.)

Owing to the anatomical position and special function of the organ the symptoms of disease of the larynx are very striking, pointing at once to the seat of trouble. Laryngeal affections are not likely to be mistaken for disease of contiguous parts, although retropharyngeal abscess, abscess at the side of the pharynx, disease of the thyroid gland, and inflammation of the lymphatics or cellular tissue in the neck may cause symptoms suggestive of laryngeal disease.

Finally morbid processes in the larynx determined by the symptoms and physical appearances may be symptomatic of the following general processes : acute inflammation, erysipelas, typhoid fever, smallpox, or measles ; chronic inflammation or ulceration, of the rheumatic or gouty diathesis, syphilis, or tuberculosis ; scars, of syphilis ; ankylosis, of rheumatic gout. The laryngeal symptoms of brain disease and of affections of the nerve-trunk have been referred to.

The practical point of all this is that affections of the larynx are not due to *primary* disease of that organ alone, but are often *secondary* either to general processes or to local morbid processes elsewhere. Therefore when laryngeal symptoms or lesions are observed, seek beyond the larynx, as well as in it, for their cause.

Diagnosis. It is seen by the above that facts obtained in the *historical diagnosis* rather suggest the cause of the affection than the disease itself.

The *subjective symptoms* do not aid much in the differential diagnosis. The value of pain, of dyspnœa, dysphonia, and cough are considered in the chapters devoted to the discussion of these symptoms.

The *objective symptom* is of the greatest value and particularly enables one to distinguish the various forms of paralysis, ulcerations, and morbid growths.

The *laboratory diagnosis* aids us to recognize the various infections, especially diphtheria.

Perichondritis.

Inflammation about the cartilages, or perichondritis, is usually phlegmonous in character, and leads to the formation of abscess. The collateral œdema is so great as to cause some obstruction, with cough and hoarseness. On palpation the larynx is extremely tender. The pain is increased by movement of the larynx, as in speaking or swallowing. If the inflammation involves the arytenoid cartilages, pain extends toward the ear, the vestibule is swollen, and the cartilage fixed. On the other hand, when the cricoid is diseased, the swallowing of solid food is painful on account of interference with the muscular attachments, there is dyspnœa, and the posterior crico-arytenoid muscles are paralyzed.

Suppurative inflammation of the thyroid cartilage may rupture externally or internally. In the latter case the abscess can be seen in the larynx. The discharge of pus and necrosed cartilage confirms the diagnosis. The bare cartilage can be felt with a probe, giving further proof of the presence of the disease. The pain may extend to the ears in carcinoma; it is propagated by the auricular branches of the vagus.

Laryngismus Stridulus.

In this form of dyspnœa the act of breathing ceases midway in an inspiration, and is attended by a characteristic sound. The disorder is seen usually in poorly nourished children. It is of frequent occurrence in *rickets*, its presence suggesting that disease when other manifestations of it are obscure. The symptoms occur suddenly and are very alarming. The child awakes in the night, and after a few short whistling inspirations suddenly stops breathing. Terror is depicted on the countenance; the eyes stare; the face is pallid at first, but rapidly becomes livid. The *alæ nasi* are extended, the head is thrown back, and the spine arched. A cold perspiration breaks out over the forehead. Carpopedal spasms may occur and urine and feces be discharged involuntarily. In a few seconds, or, at most, two minutes, the child draws two or more deep, noisy inspirations, successively lessening in depth and sound, when color returns to the face, the cyanosis gradually disappears, and the child becomes tranquil. In mild forms the child "catches its breath." It holds its breath, and then makes a noisy inspiration.

Attacks of laryngismus stridulus are more rare in adults. They may occur in hysterical subjects. A series of long, harsh, whistling or stridulous inspirations, followed by short, noisy expirations, occur during the attack. Rarely is there complete closure of the glottis.

In both children and adults general convulsions may occur during the attack, or carpopedal spasms alone may be seen. In adults the convulsions occur only in hysterical subjects.

Diagnosis. The diagnosis of laryngismus stridulus is based upon the absence of laryngeal symptoms prior to the attack, the absence of cough or hoarseness, and complete disappearance of all laryngeal symptoms

when the attack subsides. The absence of pain and fever and of laryngoscopic signs is noteworthy. This applies, of course, to spasm occurring independently of laryngeal disease.

Inflammation of the Epiglottis.

When the epiglottis is the seat of acute inflammation, there is great dysphagia on account of pain or on account of the obstruction. The sensation of a lump in the throat at the base of the tongue or the top of the larynx is complained of, and there is pain on swallowing. The pain becomes very intense at times. Fluids can not be taken, for the fluid enters the larynx when the patient attempts to swallow, because the epiglottis fails to perform its function. The voice is usually clear throughout the attack, and the general symptoms are not marked.

When the epiglottis is fixed or ulcerated, as also in some forms of ulceration of the larynx, food enters the larynx and produces dysphagia.

Acute Laryngitis.

Acute laryngitis is an inflammation of the larynx characterized by a sensation of fulness and dryness, with cough, hoarseness, and at times dyspnoea. Several varieties are observed: (1) simple acute laryngitis, (2) laryngitis with great stenosis, (3) laryngitis with membrane (laryngeal diphtheria), (4) laryngitis with spasm.

Simple Acute Laryngitis. The causes are exposure to cold, the inhalation of acrid vapors. Over-strain, as in singers or excessive use of the voice, particularly in the cold air. It may be symptomatic of the eruptive fevers, as measles, smallpox, or of erysipelas. Its occurrence in the course of chronic diseases must be looked upon with alarm, particularly in cases of Bright's disease, if dropsy is present in other situations.

The attack begins with a feeling of chilliness, followed by fever of varying degree, but usually mild. The patient complains of a feeling of pressure and dryness in the larynx, or as if a foreign body were present. Some pain gradually develops at the height of the attack, but is never so severe as to require an anodyne. From the first there is cough, dry, hacking and slightly painful. In the more intense forms the cough is continuous, disturbing the patient night and day. Paroxysms occur when the patient speaks, or takes food. First the cough is dry; within a short time it becomes moist, and expectoration of clear, transparent mucus takes place. The mucus may be tinged with blood. At the end of forty-eight hours the expectoration becomes more yellowish and opaque. The voice may be merely hoarse, or may be lost entirely. Sometimes *aphonia* without general symptoms occurs in acute laryngitis.

In *laryngitis sicca*, cough and dyspnoea occur in paroxysms and are not relieved until a dry secretion is coughed up. The paroxysms take place at night or in the early morning, and may cause retching and vomiting. It is seen in adults.

Acute Laryngitis with Stenosis. No doubt some of the cases of so-called membranous croup in children are cases of acute laryngitis,

with swelling and occlusion of the glottis from congestion and the presence of tough secretion. Œdema may or may not be present. The attack begins with catarrhal symptoms. The child is languid, refuses to eat, is thirsty, and has some chilliness and rise of temperature. With the slight cough, which may be shrill, there are hoarseness and some difficulty in breathing, but no pain on swallowing. On the second day, or after the lapse of four or five days, during which time mild fever continues, the catarrhal symptoms become more marked. The voice is more hoarse or may be suppressed. The harsh, clanging cough becomes toneless, and soon the sound is suppressed. Dyspnœa is most severe, and the breathing is hurried and noisy, attended by loud whistling inspiration and snoring expiration. The stenosis is inspiratory, and may become very intense during the day or in the succeeding twenty-four hours. It is attended with violent efforts at breathing and the occurrence of cyanosis in its most aggravated form. The larynx moves up and down; the head is thrown back. There is recession at the root of the neck, along the margins of the ribs, and at the epigastrium. The lower portion of the sternum may be drawn in. Duskiness of the extremities and of the lips is observed as the stenosis becomes more marked, and finally deepens into cyanosis. It may be relieved from time to time by removal of the obstruction, by cough, vomiting, or change of position. Another paroxysm soon comes on, and with each paroxysm the lividity becomes more and more marked, the respirations continue hurried. The face becomes pale, the extremities cold, and a cold sweat bathes the brow. Restlessness is characteristic. The child tosses about in the bed or from the bed to the arms of the nurse. The heart's action increases in frequency and becomes weaker each hour as the stenosis advances. As exhaustion ensues and the symptoms of obstruction become more marked, stupor deepening into unconsciousness develops. Convulsions may occur at the end. The attacks rarely recur if the patient once recovers. They follow exposure to cold.

If recovery takes place, the dyspnœa gradually subsides, the cyanosis fades, and the restlessness disappears. Relief is followed by a prolonged sleep, but the voice may remain hoarse or suppressed, and the cough continue many days.

Laryngeal Diphtheria. The same symptoms are seen in membranous croup and laryngeal diphtheria. In the latter affection there may be a history of exposure or of infection. At the commencement of the attack the diphtheritic patches may be seen in the fauces or nares. If a membrane can be secured and a bacteriological examination made, the diagnosis of diphtheria with stenosis is positive. Enlarged glands in the neck, with marked physical depression, a moderate degree or entire absence of fever, and the occurrence of early albuminuria, also point to diphtheria. The distinction between the two affections is nevertheless quite difficult, and as long as there is a shadow of doubt, the case should, for prophylactic reasons, be considered one of diphtheria.

Acute Laryngitis, with Spasm—False Croup or Spasmodic Laryngitis. Another form of laryngitis associated with spasm of the larynx is seen in children. The catarrhal symptoms are mild, so that the child

seems to be well during the day. Fever is absent, and a slight cough or huskiness alone calls attention to the larynx. After the first three or four hours of quiet sleep the child suddenly awakes with a barking cough, sits up and struggles for breath. The dyspnœa continues from a few minutes to an hour or so, gradually lessening, to disappear entirely as the child lapses into sleep. Throughout the next day the child seems as well as on the previous day, and the succeeding night is seized with another attack of "croup." This may occur once or twice during the night. It seems to be influenced by the weather. Damp days and an east wind are provocative of an attack. It recurs frequently during the same season.

Œdema of the Larynx. This condition develops in the course of acute laryngitis, quite frequently in chronic diseases of the larynx, particularly if ulceration is present, and as a complication of erysipelas and diphtheria. In some cases of Bright's disease it may develop suddenly.

Symptoms of laryngeal stenosis may occur suddenly. The voice becomes husky and suppressed, the dyspnœa is very extreme so that in a few hours grave symptoms of obstruction arise. There is no cough. The patient complains of the sensation of a foreign body, and tries to grasp it.

Acute Submucous Laryngitis. The inflammation extends to the submucous cellular tissue. It arises in the course of acute laryngitis, and is the form seen in traumatism, or from burns and scalds. The symptoms are those of intense laryngitis, with stridor. They increase in severity until stenosis arises. If the lower surface of the cords is affected, death occurs from asphyxia. Sometimes the inflammation is circumscribed and is followed by development of an abscess.

The *chronic form of submucous inflammation* of the larynx is usually seen in drunkards, and is recognized usually by the laryngoscopic examination. The symptoms are those of slight stenosis.

DIAGNOSIS. Acute affections of the larynx are distinguished from other diseases without much difficulty. To recognize the various forms of acute laryngitis, however, is not easy. In all there is a certain degree of laryngeal stenosis, and practically the question to be answered is, Which form of stenosis is present? The accompanying table shows the differential points of diagnosis. It is seen that the age, the occurrence of previous attacks, the character of the general symptoms, the history of previous laryngeal disease, the association of faucial disease, the presence or absence of membrane, and the results of laryngoscopic examination must be considered before making a positive diagnosis.

<i>Simple Acute Laryngitis—"Catarrh of Larynx."</i>	<i>Acute Laryngitis with Stenosis.</i>
Gradual onset of laryngitis, with dyspnœa very slight or absent.	Gradual onset of laryngitis, but dyspnœa develops to great severity.
All ages.	Children.
Fever of varying degree.	Fever of varying degree.
Dry irritating cough.	Dry cough, often paroxysmal.
May be hoarseness.	Hoarseness.
Pharynx reddened.	Pharynx reddened.
Gradual increase and decline.	Gradual increase, and either death of patient or decline of dyspnœa.
Larynx red and slightly swollen, as seen by laryngoscope.	Same, but swelling much greater.

Acute Laryngitis with Spasm—Spasmodic Croup.

May be slight hoarseness or cough, or none.
Suddenly, in night, child awakes with intense dyspnoea and crowing inspiration.

Children.

Temporary high fever.

Slight brassy cough during day.

May be slight hoarseness in day. Very hoarse in attack.

Lasts a few minutes to one hour. May recur, or no attack until next night.

Slight redness, or nothing seen by laryngoscope.

Edema of Larynx.

Some inflammatory disease of larynx exists.
Rapid development of dyspnoea, increasing to great severity.

All ages.

Depends on cause.

No cough.

No hoarseness.

Increases steadily to climax, then death, or decline of dyspnoea.

Epiglottis and aryteno-epiglottic folds swollen, pale, and waxy.

Foreign Bodies.

During eating or while holding object in mouth, sudden dyspnoea, varying in intensity according to object.

All ages.

No fever.

Irritative, expulsive cough.

May be hoarseness or not.

Cough persists till removal of body, or occasionally the larynx becomes accustomed to its presence, and cough ceases.

See the foreign body.

Laryngismus Stridulus—"Child-crowing."

No laryngitis. Sudden attacks of dyspnoea with crowing inspiration, either day or night. Very severe. May be general convulsions.

Children or hysterical adults.

No fever.

No cough.

No hoarseness.

Occurs often in rhaehitic and hysterical cases.

Ends suddenly, in at most two minutes, and recurs often.

Nothing seen in larynx.

Membranous Laryngitis—Croup; Diphtheria.

Epidemic.

Gradually developing hoarseness and croupy cough, with low fever and lassitude, then development of dyspnoea, gradually and without intermission, as a rule.

Children.

Low fever and depression.

Croupy cough, later suppressed.

Very hoarse.

Fauces red and often with membrane; albuminuria; paralysis.

Increases steadily, broken by intense paroxysms. Either death or gradual improvement.

Red, swollen, with membrane.

Pertussis—Whooping-cough.

Epidemic.

Bronchitis, with cough developing in from one to three weeks. Then dyspnoea caused by severe paroxysms of coughing—absent between them.

Children.

Only the fever due to bronchitis.

Intense paroxysm of coughing.

No hoarseness.

Hemorrhages in various places from strain or emphysema.

May be death from exhaustion, or gradual improvement.

Nothing seen, unless slight laryngitis.

Chronic Laryngitis.

Chronic hoarseness may be due to chronic laryngitis. This affection either originates in an acute attack or comes on slowly. Prolonged use of the voice in a higher key than natural or in the open air, the abuse of alcohol, and constant exposure, are exciting causes. It may be symptomatic of syphilis or tuberculosis, and frequently results from inflammation of the upper air-passages, and particularly from chronic pharyngitis. It occurs more frequently after middle life, and usually in the male sex.

Prolonged or excessive use of the voice gives rise to discomfort with dryness and tickling in the throat. At first the secretion of mucus is very slight, but after hawking and coughing it increases in amount. Hoarseness occurs, and if the patient is careless or persists in the harmful occupation, complete aphonia may result. The voice is clearest in the morning, after expectoration of the nocturnal mucus, but becomes husky toward night. The aphonia may occur in paroxysms, and is relieved by coughing up a dry secretion. The cough is never severe. The sputum is small in amount, glairy, often in little balls or crusts.

Paralyses of the Laryngeal Muscles.

They are divided for convenience into groups. The *symptom* is dysphonia, which, with laryngoscopic appearances, leads to the recognition of the particular form of the paralysis.

1. Paralysis of the Tensors of the Cord. The cricothyroid muscle is paralyzed; the superior laryngeal nerve which supplies the muscle is affected. The *voice* is deep and rough, and incapable of producing high tones. Usually the whole nerve is involved, and the result is *anæsthesia* of the larynx and *paralysis of the epiglottis*.

LARYNGEAL EXAMINATION. The *epiglottis* is fixed, and falls back against the tongue. The *glottis* forms a wavy line.

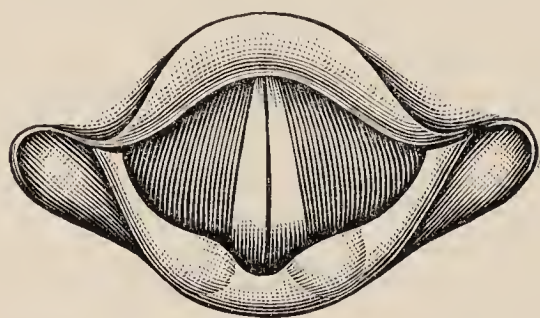
CAUSAL DISEASE. The condition occurs almost exclusively after diphtheria.

2. Paralysis of the Closers of the Glottis, or Adductors of the Cords. The muscles involved are the crico-arytenoideus lateralis, arytenoideus transversus, and the thyro-arytenoideus internus and externus. The nerve is the recurrent laryngeal.

The symptoms are *complete aphonia*, coming on suddenly, and often disappearing as suddenly.

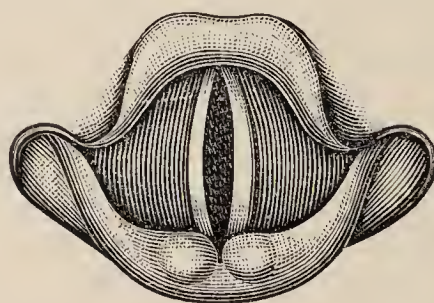
LARYNGEAL EXAMINATION. During phonation the cords remain in the inspiratory position. The paralysis may affect one or both sides.

FIG. 341.



Paralysis of the arytenoideus transversus in phonation. (GOTTSTEIN.)

FIG. 342.



Paralysis of the thyro-arytenoideus internus in phonation. (GOTTSTEIN.)

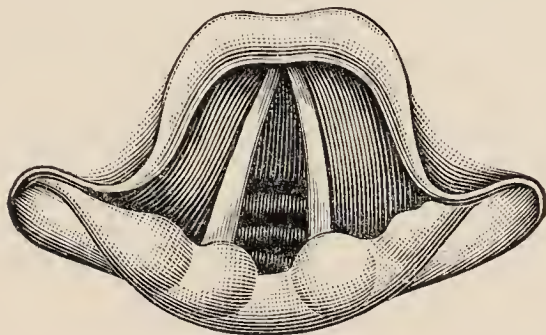
Sometimes the arytenoideus transversus alone is affected. Then there is hoarseness or aphonia. The anterior portions of the cords come together in phonation, but the posterior portions do not, leaving a triangular opening posteriorly. (See Fig. 341.) In other cases the thyro-arytenoideus internus alone is affected. There is then dysphonia or aphonia, as before, but the cords come together at both extremities and remain apart in the middle, forming an oval opening. (See Fig. 342.)

CAUSAL DISEASE. These paralyse occur in hysteria, catarrh, or severe over-strain of the voice.

3. Paralysis of the Openers of the Glottis, or Abductors of the Cords. The muscle affected is the crico-arytenoideus posticus; the nerve is the recurrent laryngeal.

SYMPTOMS. When one side is affected, the respiration is free, but there is stridor or forced inspiration. The voice is harsh.

FIG. 343.



Paralysis of the left recurrent nerve; inspiration. (GOTTSTEIN.)

LARYNGEAL EXAMINATION. One cord remains in the median line. (See Fig. 343.) When both sides are affected, inspiratory dyspnoea with stridor gradually develops. The voice is nearly normal. The glottis forms a narrow cleft which becomes still narrower on inspection.

4. Complete Paralysis of the Recurrent Laryngeal Nerve. Unilateral Paralysis. A weak, toneless voice which breaks into a falsetto when the patient endeavors to speak loud.

LARYNGEAL EXAMINATION. The cord and arytenoid body are in the cadaveric position, viz., half-way between the phonating and the inspiratory positions. In phonation the other cord passes beyond the median line, and the glottis is slanting. The edge of the paralyzed cord is excavated.

Bilateral Paralysis. Aphonia and inability to cough and expectorate.

LARYNGEAL EXAMINATION. Both cords are in the cadaveric position and their edges excavated. The adductors are usually paralyzed before the abductors, and one can see all the intermediate stages by close observation.

CAUSAL DISEASE. The conditions that give rise to the paralysis are numerous. It may arise from simple catarrh or from hysteria. More often it is due to pressure on the vagus or recurrent laryngeal, or some disease affecting these nerves or their roots.

The causes of pressure are: aneurism of the subclavian or aorta, mediastinal tumor, tuberculous bronchial glands, the apex of a tuberculous lung, cancer of the œsophagus, goitre, and carcinoma of the pleura.

The diseases are: diphtheria, tumor, softening or hemorrhage into the brain, bulbar paralysis, neuritis, typhus, cholera, variola, articular rheumatism, toxæmia (?), sclerosis of the cord, progressive muscular atrophy, and paralytic dementia.

Tumors of the Larynx.

Both benign and malignant growths are seen. At first *dysphonia* or *aphonia* takes place. The impairment of voice may continue for a long period of time before *dyspnœa* arises. This develops very gradually, and in some few cases is attended by an irritative *cough*. The general symptoms are not marked in benign cases. In the malignant forms they are pronounced, but characterized by the development of cachexia later than in carcinoma elsewhere.

The *diagnosis* of malignant disease of the larynx is based upon the association of symptoms of laryngeal disease with pain, and with the characteristic appearances found on inspection, on its occurring after the middle period of life and lasting only from six to nine months with the development of cachexia and emaciation without fever. Enlargement of the cervical glands points to cancer. Simple and syphilitic perichondritis must be excluded.

Tuberculosis of the Larynx.

The occurrence of primary laryngeal tuberculosis is doubtful. It can not be proved clinically, and the majority of cases at least are secondary to tuberculosis of the lungs. The manifestations of tuberculosis of the larynx may be either a simple persistent catarrh, an infiltration, or an ulceration. (See pages 443 and 444.) The symptoms vary according to the lesion:

a. Catarrh. There is a slight hoarseness and the voice tires easily. Often paræsthesia or peculiar sensations in the larynx are present. Cough, when due to this alone and not to the process in the lungs, is short and dry.

b. Infiltration. At first the symptoms are those of simple catarrh; then the alteration of the voice increases even to aphonia; there is a feeling of dryness or soreness in the larynx, with dysphagia. The cough is very slight and is usually wholly disguised by the cough due to the disease in the lungs. There is some difficulty in expectoration.

c. Ulceration. The symptoms are the same as those of infiltration, but the dysphagia and pain are greater.

Diagnosis. Tuberculous ulcer occurs most frequently in the male sex, and during the period ranging from eighteen to thirty years of age. If the symptoms develop in the course of phthisis, or in case that affection can not be recognized, if there is a history of infection or exposure, and if bacilli are found in the sputum, the diagnosis is not difficult. A portion of the diseased mass may be removed for microscopical examination or inoculation. In examining the secretion for tubercle bacilli, it is to be remembered that the exudate may have been brought up from the lungs. The examination in cases of phthisis is of little practical value, except to determine whether the ulceration present may be syphilitic and grafted upon a tuberculous disease of the lungs. Enlargement of the glands of the neck is often present, but is not diagnostic.

Fever is present, and, indeed, may be an important diagnostic feature in doubtful cases. The temperature should be taken every two hours,

for the morning or evening exacerbations may not be present. Emaciation ensues, and sooner or later the hectic phenomena and signs of tuberculosis in other structures arise. When tuberculous ulceration of the larynx occurs in the course of local pulmonary tuberculosis, the disease runs a much more rapid course.

The *laryngeal symptoms* are not diagnostic. Pain may be the most distinct. The laryngoscopic findings are more characteristic. Local anæmia with paræsthesia, paresis of the cords, and short cough, and an obstinate diffuse catarrh are suspicious symptoms. The peculiar ridged infiltration between the arytenoids is almost invariably tuberculous.

Isolated thickenings anywhere in the larynx, tapering gradually into the normal tissue, can only be tuberculous or syphilitic. The regularity and number of the lesions, with anæmia and absence of inflammatory signs, usually suffice to distinguish the tuberculous from the syphilitic form. The ulcers are non-erosive. Syphilitic ulcers do not often occur anywhere but on the edge and lingual side of the epiglottis and on the cords. They extend more rapidly than the tuberculous, and may be continuous with ulceration in the pharynx. The area of ulceration may extend to the base of the tongue, which is very frequent in tuberculous disease. In syphilitic ulceration scars or cicatrices are seen, but they are absent in the tuberculous form. Laryngoscopic examination in tuberculous ulceration is difficult, as it causes great pain; in syphilis comparatively little pain attends examination. (See the Infections.)

Syphilitic Affections of the Larynx.

Mucous patches, papules, infiltrations, or gummata may be present in the larynx for some time without causing symptoms. Usually a change in the voice is the first symptom noticed, due either to the catarrh or to ulcers, scars, infiltrations, or gummata affecting the cords. There is often a feeling of pressure or a tickling sensation. Pain is not usual, and when present is very slight. Dysphagia occurs only when the epiglottis is extensively ulcerated. There is little or no cough.

The *diagnosis* rests upon the history of infection, the objective signs of syphilis indicated by pigmentation or recent eruption, scars, periostitis or nodes on the bone, and enlarged glands. The laryngeal symptoms are not diagnostic, save that pain is absent in spite of extensive ulceration, while difficulty of deglutition, on account of food entering the larynx, is of frequent occurrence. The laryngoscopic appearances, as indicated above, are characteristic. In obscure cases the distinctions spoken of in tuberculosis are of diagnostic value.

Although the patient may be broken down and cachectic, the febrile range is not high, unless perichondritis occurs, or pneumonia sets in on account of food in the air-passages.

The Larynx in Other Diseases.

Laryngeal symptoms due to lesions of the nervous system are found under the following circumstances. (See Cerebral Localization.)

Cerebral Hemorrhage. 1. **Aphasia.** The movement of the muscles is normal, but they can not be controlled by the will. It is caused by hemorrhage in the cortex or along the course of connective fibres.

2. **Recurrent paralysis.** Due to hemorrhage in the medulla.

3. **Symptoms of bulbar paralysis.** Same cause.

Encephalomalacia.. (Softening.) When in the brain, aphasias result; when in the medulla, bulbar symptoms.

Tumors of Cerebrum. The symptoms are, according to location, aphonia, aphasia, or paralysis of the cords.

Bulbar Paralysis. We have, of course, the other symptoms of the disease. The voice becomes weak and monotonous, devoid of modulation. High tones are impossible. The disturbance progresses to hoarseness and finally aphonia. Particles of food and drink enter the larynx. Paresis or paralysis of the cords results.

Multiple Sclerosis. The speech is low, uncertain and scanning, later hoarse. Laughing and crying are accompanied by peculiar yawning inspirations. On laryngoscopical examination slight paresis of the cords is seen.

Posterior Sclerosis (*Tabes*). The muscles act very slowly. Sometimes symptoms of irritation, as tickling or burning in the larynx, with a dry cough, occasionally severe paroxysms of coughing, even to spasm of the larynx, occur—"laryngeal crises." In rare cases a phonetic spasm has been observed. Less often paresis or paralyzes of the various muscles occur, most frequently the posticus, next the recurrent. Sensibility may or may not be disturbed.

Amyotrophic Lateral Sclerosis. There is a mixture of bulbar with spinal symptoms. (See Sclerosis.)

Progressive Muscular Atrophy. The same mixture of symptoms occurs very late.

Paralytic Dementia. There may be disturbances in articulation, with paresis and paralysis of the cords.

Chorea. There may be a tremor of the cords from under-tension, but probably no true choreic movements.

CHAPTER VI.

DISEASES OF THE LUNGS AND PLEURA.

Pathology. Disease of the lungs may be due to alterations in (*a*) the connective tissue, (*b*) the channels, (*c*) the muscles, and (*d*) the nervous mechanism. The pleura is subject to diseases common to serous membranes.

Alterations in the Connective Tissue. The connective tissue, composing the structure of the lungs independently of the canals, although liable to the same morbid processes that affect it in other situations, is not often subjected to irritants that cause acute inflammation. Its chronic inflammations, in the large majority of cases, occur secondarily to processes in the channels.

Alterations in the Channels. Diseases of the lungs are usually diseases of their channels, due to morbid alterations of them (1) by processes common to the structure of such channels and (2) by obstruction of them.

1. MORBID PROCESSES OF THE CHANNELS AND THE SYMPTOMS PRODUCED. The *air-tubes* are lined with mucous membrane, which is subject to the morbid processes that attack such lining: congestion or acute and chronic inflammation. A flux is the characteristic symptom. The muscle and the elastic tissue of the canal become involved in the process. The former undergoes spasm, with or without inflammation of the mucous membrane (asthma). Grave consequences do not arise until degeneration takes place, with loss of the power of confining the air or driving it out and resulting emphysema. In the *blood-canal*, *hyperæmia* (congestion), *embolism* and *thrombosis*, and secondary *œdema* may take place; in the *lymph-channels*, *inflammation* (acute and chronic pleurisy), and *transudation* (hydrothorax or hæmothorax) occur. The *symptoms* that arise in each or all of the above processes—pain, local discomfort, mucous or purulent discharge, serous or purulent exudation and fever—are not different from those which are found in diseases of similar tissues in other localities. (Compare with affections of mucous or serous membranes in other organs.)

2. OBSTRUCTION OF THE CHANNELS AND THE SYMPTOMS PRODUCED. In addition to these, there is a group of symptoms due to obstruction of the various channels, causing interference with the function of the lungs. The symptoms are purely mechanical. Obstruction of either the *bronchial tubes* or *bloodvessels* gives rise to *dyspnœa*. This is as pronounced in asthma or capillary bronchitis as it is in embolic obstruction (fat-embolism) or congestion of and stasis in the bloodvessels. It occurs when the canals are occluded by extrinsic causes—the presence of foreign bodies in the bronchi or the presence of pleural effusions. As a sequence we

have another vivid picture—the development of *cyanosis* from interference with aëration.

Alterations in the Muscles and the Symptoms Produced. *Inactivity* of the muscles, from *pain*, from *debility*, or from *paralysis* through any cause practically occludes the canals by lessening the movement of their normal contents. The amount of air therefore is diminished and dyspnœa results.

Alterations in the Nervous Mechanism and the Symptoms Produced. Various forms may exist. (a) A *central affection*, causing pulmonic symptoms from the following causes: 1. The influence of the higher centres upon the lower pulmonary centre, as seen in hysterical cough, or emotional cough, and in asthma—*respiratory neuroses*. 2. *Disease* affecting the region of the centre, as in *tumor* or in *bulbar* or glosso-labio-laryngeal *paralysis*. 3. *Irritants* acting upon the centre, as *urea*, exciting uræmic asthma. (b) An *affection* of the *nerve-trunk*, as from the pressure of an aneurism or morbid growth. (c) *Reflex influences* through the pneumogastric and correlated nerves. The asthma of nasal disease, or of peripheral irritation, and reflex cough (*neuroses*) are of this nature.

Differential Diagnosis. It is not usually difficult to distinguish diseases of the lungs from affections of other structures. Pleurisy and pleurodynia, it is true, are often differentiated with difficulty. We may be called upon to decide between pleurisy and subdiaphragmatic inflammation, between a pleural and an hepatic inflammation, between a pleuritis and a pericarditis and between cardiac and pulmonary disease, especially when in the presence of both conditions we desire to determine which is primary.

Chronic affections of the lungs, of the mediastinum, and of the great vessels must be distinguished from one another; an aneurism or mediastinal disease, from chronic phthisis. With care in ascertaining the history and the subjective and objective symptoms the distinction may not be difficult.

It often happens in pulmonary disease that some of the pronounced symptoms may point strongly to an infection other than that of the lungs; thus the cerebral symptoms of pneumonia may be attributed to meningitis, or the fever present thought to be due to typhoid fever. On the other hand, the presence of a pulmonary affection, as tuberculosis, may explain the nature of a morbid process in other organs or structures. Hence, in all cases in which there is a possibility of secondary tuberculosis the lungs should be examined in order to determine if they are the seat of the primary disease. In this way the true nature of a meningitis, of a peritonitis, or of another tubercular affection may be recognized. In secondary anæmia and in protracted debility of unknown source the lungs always should be examined. It must be borne in mind that in chronic diseases, such as chronic renal disease, chronic arthritis, diabetes, etc., pulmonary tuberculosis may set in most insidiously. In the same diseases, pneumonia frequently is a terminal infection, and likewise runs an insidious course. In the extremes of life pulmonary infections, such as pneumonia, present symptoms out of the usual run. In infancy and childhood the cerebral symptoms may mask the pulmonary

symptoms ; in senility the absence of cough or expectoration may lead to the dismissal of all thought of pulmonary disease. In short, the lungs should be examined in all affections.

This injunction is to be observed particularly inasmuch as lung diseases are often secondary to other diseases : phthisis to tuberculosis elsewhere : pneumonia or pleurisy to all infectious disorders, to Bright's disease, cancer, and diabetes. The possibility of a hydrothorax, secondary to causes of transudation, must always be borne in mind.

Relative Value of Subjective and Objective Symptoms. The subjective symptoms are few, and are common to so many pulmonary diseases that they are of little diagnostic value. The objective symptoms obtained by applying the laws of physics to the lungs are of greater service. The effect of the occlusion of the channels is mechanical or physical ; hence a physical change in the lung follows. The objective symptoms occur when the air-movement is checked and no sounds are heard ; when abnormal breath-sounds and new sounds (*râles*) are created ; and when air is replaced by solid structure. The objective signs of these conditions are determined by inspection, palpation, percussion, and auscultation.

DISEASES OF THE LUNGS AND BRONCHI.

Classification. Affections of the lungs may be divided into (*A*) the neuroses, (*B*) the congestions, (*C*) the inflammations, (*D*) the degenerations, (*E*) the morbid growths, and (*F*) those due to gross parasitic invasion. Influences operating through the pneumogastric and phrenic nerves may be responsible for respiratory neuroses. The congestions are so intimately associated with vascular phenomena that the latter may be included in the process. The inflammations are limited to the bronchi, to the alveoli, and to the connective tissues surrounding both. The intimate relation of the small bronchi, the alveoli, and their surrounding connective tissues implies their conjoint involvement in many processes.

A. The Neuroses.

B. The Congestions.

1. Active, including *hemorrhagic* infarct.

2. Passive.

Subsidiary : *hemorrhage*.

C. The Inflammations, chiefly *infections*.

1. The Bronchi.

Acute.

Chronic.

2. Bronchi and alveoli.

Bronchopneumonia (an infection).

3. Bronchi, alveoli, and connective tissue.

Pneumonia.

Tuberculosis.

Abscess of the lung.

Gangrene.

Chronic interstitial pneumonia—pneumonokoniosis.

Syphilis of the lung.

D. The Degenerations.

Emphysema.

Bronchial dilatation.

E. Morbid Growths.

F. Gross Parasites.

Hydatid disease.

THE NEUROSES.

The neuroses are affections of the lungs unattended by structural change. To this class belong the varieties of rapid breathing, of slow breathing, of cough and of dyspnœa which appear to arise without structural change, and which are discussed exhaustively in the section devoted to the subjective symptoms. Of the neuroses, *asthma* is the most common form. Reference need not be made further to the respiratory neuroses, but it should be borne in mind that their presence may or may not be unattended by organic change in the lungs. On the other hand, we are likely to find the general phenomena or stigmata which are associated with neuroses of other organs, as well as the lungs. Hence, the condition of neurasthenia is likely to be present on the one hand, or the numerous stigmata of hysteria may be found on the other.

Asthma.

Asthma is a chronic disease caused by spasmodic narrowing of the bronchial tubes, and characterized by paroxysmal attacks of dyspnœa, diminished respiratory movement of the chest, and prolonged expiration, attended by a wheezing sound and sibilant râles, and which end abruptly with the expectoration of tenacious mucus. The attack may be limited to a single night, or may be prolonged for days, with nocturnal exacerbations.

Symptoms. Premonitory symptoms are said to occur in about one-half the cases. These are for the most part nervous, such as headache, neuralgia, irritability of temper, vertigo, and drowsiness. Hyde Salter found that there were premonitory symptoms in 111 out of 226 cases collected by him. In 63 they were nervous, in 8 they consisted of profuse diuresis, and in 14 they were connected with the digestive system.

The attack itself usually begins during sleep, and often at a regular time. It may, however, begin during the day, and at a certain hour, independently of sleep. The onset is manifested by tightness across the chest and more or less difficulty in breathing. This dyspnœa increases rapidly and often reaches an extreme degree. The face becomes pale and anxious, and may be covered with a cold perspiration; the lips are dusky from insufficient oxygenation of the blood. The patient feels smothered, and makes frantic efforts to get his breath, rushing to an open window, no matter how cold the weather, or, if unable to leave the bed, sitting up with the hands pressed upon the bed so as to give purchase to the accessory muscles of respiration. Notwithstanding the great respiratory efforts made, the chest moves but little, because the lungs are already distended to the extent of a full inspiration. The patient is unable to expel the contained air, owing to the spasm of the bronchial tubes.

The frequency of respiration is diminished, sometimes to one-half the normal; the rhythm is also altered, inspiration being short and gasping, and followed without pause by expiration, which is much prolonged and accompanied by a wheezing sound audible to bystanders.

There is an increased amount of air in the thorax, and inability to remove it. The duration of an attack of asthma varies from half an hour to a day or two. In patients with chronic bronchitis it may be prolonged for a week or two, with remissions during the day. It may subside abruptly or by degrees.

Subsidence of an attack is marked by expectoration, the sputa having special characteristics. (See under Sputum.) At first it is made up of rounded gelatinous masses, which when unfolded in water are seen to be made of spirals. Later it becomes mucopurulent. Curschmann's spirals and the Charcot-Leyden crystals are nearly always found. The leucocytes are increased, and 25 per cent. of them are eosinophiles.

The causative factors in asthma are various. About twice as many males as females are affected, and there is a marked hereditary tendency in some families. There is probably some special peculiarity in asthmatic patients, but just what it is, has not been determined. It may reside in the lungs, and may be part of a general constitutional irritability. (Salter.) Bronchitis, emphysema, and heart disease act as causes, and also syphilis, malarial poisoning, and chronic Bright's disease.

The above description applies to spasmodic asthma, the dyspnœa up to this time having been paroxysmal. Sooner or later it becomes constant, and then other changes take place in the lungs. First, there is persistent bronchitis; second, the presence of emphysema. Indeed in many cases it is difficult to ascertain the exact sequence of affections. In emphysema of the lungs, dyspnœa is constant, but on exposure to cold or on account of an attack of indigestion more severe paroxysms may occur, as well as asthmatic attacks, although the patient is not an asthmatic. On the other hand, a patient may have had asthma for a number of years, during which attacks of dyspnœa occurred only in paroxysms. As time passes, the paroxysms become more and more frequent, and emphysema develops. With the advent of emphysema the dyspnœa becomes more constant.

Physical Signs. On *inspection* the chest is enlarged, barrel-shaped, its movement is lessened and strikingly out of proportion to the muscular exertions. The diaphragm is lowered.

On *percussion* hyper-resonance is elicited; on *auscultation* expiration is faint and short, and inspiration is prolonged, and sibilant and sonorous râles are heard, more marked on expiration.

THE CONGESTIONS.

Active Congestion of the Lungs.

In active congestion there is an increased amount of blood, which diminishes the air-space by encroachment and causes more or less consolidation.

Symptoms. Dyspnœa, cough, and frothy, bloody expectoration attend

the fluxion. No cases have yet been reported in which bacteriological examination of the sputum was made. Of course, *Micrococcus lanceolatus* is not found in the sputum.

Physical Signs. Increased fremitus, impaired resonance or dulness, and bronchial breathing are present. They are observed on both sides, usually at the bases.

Diagnosis. If the above signs and symptoms develop suddenly—within twenty-four hours—a fluxion to the lung has in all probability taken place. If the patient is subject to heart disease, or if he has been exposed to and has inhaled hot vapors or irritants, the probability of fluxion is increased. The occurrence of fever would point to pneumonia as the cause of the objective and subjective symptoms.

Passive Congestion of the Lungs.

Mechanical congestion occurs when the flow of blood to the heart is obstructed, as in organic valvular disease or insufficiency. Rarely the pressure of tumors on the pulmonary veins acts in a similar manner.

Hypostatic congestion occurs in fevers, as protracted typhoid, and in prolonged general exhaustion or adynamia. Ascites or other affections below the diaphragm, which lessen the respiratory excursion, cause this form.

Symptoms. Dyspnœa, cough, and expectoration of blood-stained sputum are common. The sputum contains alveolar cells, often pigmented, but no micro-organisms.

Physical Signs. Consolidation is present, manifesting itself by slight dulness and feeble or bronchial breathing; the bronchial mucous membrane is also congested, giving rise to abundant large râles. The affection is bilateral and usually confined to the posterior portions of the bases.

Œdema of the Lungs.

The air-cells and alveolar walls are filled with serous exudation, as in œdema of the skin. The condition is frequently due to the weakness of the heart, which occurs at the end of long-continued diseases of an exhaustive nature, particularly if the heart is over-taxed. It occurs, therefore, in the terminal stages of chronic Bright's disease, of organic heart disease, of the anæmias and cachexias. Both congestion and œdema occur in cerebral affections.

Symptoms. They are those of congestion in a more aggravated form. Dyspnœa, cough, and the expectoration of large quantities of a sero-mucoid fluid are seen. In cases of myocarditis or acute dilatation of the heart, in valvulitis with failing compensation, œdema of the lungs often takes place suddenly. It may follow some unusual exertion. Its onset is attended with more or less collapse, increased pulse-rate, hurried, oppressed, noisy breathing, cyanosis, and an anxious expression.

Physical Signs. These are an unusual number of small râles throughout the chest, apparently created in the air-sacs, and imperfect resonance, showing that some lobules are collapsed.

Pulmonary Embolism and Thrombosis.

Pulmonary embolism consists in plugging of the pulmonary artery or its branches by coagula formed in the right heart or in the veins. The symptoms depend upon the size of the occluded vessel and upon the nature of the embolus—*i. e.*, whether septic or not. If the artery itself is plugged, death takes place suddenly or after a short interval, with symptoms of syncope or asphyxia.

Plugging of a Large Branch. Symptoms. The first symptom is generally intense dyspnœa, which may amount to an agonizing craving for air. Pain in the chest, which may or may not be acute, is complained of, and may be referred to the seat of the embolus. Cough is not a common symptom, and may be altogether absent. The breathing is considerably altered; it is usually increased in frequency, and may be much hurried; it may or may not be shallow, and while the patient can take a deep inspiration, it does not give relief to his dyspnœa. At times it is irregular and gasping.

The face is pale or may be cyanosed, and is apt to be bathed in perspiration. The veins are swollen and prominent. The heart's action is irregular and may be tumultuous. Exophthalmos has been observed. The temperature falls below normal, but a febrile rise may occur later. The intellect is unclouded.

Physical Signs. These are indefinite. The respiratory murmur is roughened and exaggerated in most, but not in all cases. Fox states that râles are very rarely heard. Collapse, œdema, and bronchitis are possible results. A systolic blowing murmur may be heard over the heart and pulmonary artery, and in protracted cases albuminuria and œdema may be met with.

Septic Embolus. When the embolus is septic, a *septic pneumonia* or *metastatic abscesses* are probable results in cases not immediately fatal.

Hemorrhagic Infarcts. Symptoms. When the emboli produce hemorrhagic infarcts, the symptoms are milder, and consist principally in dyspnœa, pulmonary hemorrhage, and palpitation. The onset is sudden and accompanied by a fall in temperature.

Physical Signs. These indicate consolidation, if the pneumonia or infarcted area is of moderate size. The consolidation may be discovered at the root of the lungs in the interscapular region.

Cardiac Embolus. Hæmoptysis is a common symptom when the embolus has arisen in the heart. The amount of blood varies from a copious expectoration to the rusty sputum seen in pneumonia; it may persist for weeks. Pleurisy and pleural effusion are frequent complications; chills occur sometimes, and pneumonia, with corresponding rise of temperature, may develop.

The most important points in diagnosis are the sudden onset of the dyspnœa and other pulmonary symptoms, and the detection of a condition which would give rise to emboli, such as puerperal fever or heart disease.

THE INFLAMMATIONS.

Inflammations of the bronchi are distinguished from other diseases of the lungs chiefly by the difference in the physical signs. Except in capillary bronchitis, the general and subjective symptoms are not so severe as in other affections.

We are aided in the recognition of bronchial affections, first, by the fact that they are bilateral; second, that the bases are usually affected; third, that there is diminution of fremitus determined by palpation; fourth, that there is absence of dulness on percussion; fifth, that râles are more pronounced in proportion to other physical signs, and more general than in other lung affections.

Acute Bronchitis.

This is an acute inflammation of the mucous membrane of the larger or middle-sized bronchial tubes. It occurs most frequently by extension of the catarrhal inflammation from the nose and throat; but in some persons it develops so suddenly that it appears to be primary in the tubes. The duration of the disease is from a few days to several weeks. It is never fatal except in the very old and very young, or in those who are much debilitated.

Symptoms. The patient complains of soreness or rawness underneath the sternum, especially at its upper part. There are frequently a feeling of tickling in the throat, and a sense of weight or oppression in the chest. Chest pain is due to myalgia or the strain upon the muscles from coughing. The *cough* is at first hard and dry, and often produces pain of a tearing character in the muscles of the chest and abdomen. The cough is apt to be worse when the patient first lies down, and again on rising, especially after a night's rest. *Fever* is usually slight and of short duration. The respirations are accelerated, but not markedly, and there is no dyspnoea. The *expectoration* is at first a white, frothy, viscid mucus, subsequently becoming more abundant and mucopurulent. The cough and expectoration usually last for some time after fever has subsided.

Physical Signs. In uncomplicated cases there are no changes in the physical structure of the lungs. On examination of the chest the *percussion-note* is found to be clear; the *respiratory murmur* more roughened and harsher than normal, but not bronchovesicular or bronchial; accompanying breathing there are heard sibilant and sonorous *râles*, and in the later stages some large and medium-sized mucous râles. The râles vary in position from time to time, and especially after coughing. *Vocal resonance* and *fremitus* are unaltered. A *rhonchal fremitus* may be produced by sonorous râles.

Diagnosis. The diagnosis of acute bronchitis is easily made by noting the fact that the disease runs an acute course marked by fever, cough, and expectoration; and that the physical signs are negative, except as to roughening of the respiratory murmur and the existence of bronchial râles, heard on both sides of the chest.

From *croupous pneumonia* and *chronic local tuberculosis* of the lungs,

acute bronchitis is distinguished by the absence of dulness on percussion, of bronchial breathing, and of increase in the vocal resonance and fremitus; by the absence, in other words, of the ordinary signs of consolidation. From pneumonia it is further distinguished by the milder character of the subjective symptoms, and by the fact that physical signs are almost always bilateral, while in pneumonia they generally are unilateral. Tuberculosis is further distinguished by its slow progress and by its involvement of the apices preferably, whereas bronchitis is more marked at the bases, and by the occurrence, sooner or later, of hectic fever and emaciation, which are absent in bronchitis. Doubt will exist only at first; the progress of the case will in time make everything clear. Systematic examination of the sputum is an important diagnostic aid, and will lead to the differentiation of many cases of bronchitis from tuberculosis and from pneumonia. In infants and children especially, bronchitis is at times so rebellious to treatment that tuberculosis is suspected.

In *bronchopneumonia* (catarrhal pneumonia) there is a diffuse bronchitis associated with small areas of pneumonic consolidation. Bronchopneumonia is distinguished by having graver general symptoms and by the presence of small areas over which there are dulness on percussion and bronchial breathing, associated with the physical signs of bronchitis already noted.

Acute miliary tuberculosis of the lungs is very easily mistaken for bronchitis, because dulness, if present, amounts to nothing more than tympanitic dulness, because the signs are diffused through both lungs, and because the respiratory murmur is fainter than normal and only slightly roughened. Close inspection of the patient will, however, make it evident that his condition is worse than could be accounted for by bronchitis alone. The fever is higher, the respirations are more frequent, pallor, with a dusky or faintly cyanotic hue intermingled, is common, and perspiration is more pronounced. A primary focus or a source of infection may be discovered.

Acute bronchitis may be mistaken for *spasmodic laryngitis* (croup). It is distinguished by the fact that the spasms are less pronounced in bronchitis, and there is fever in addition to the physical signs. In bronchitis the breathing is rarely so stridulous as in laryngeal spasm.

Whooping-cough can not be distinguished positively from bronchitis before the characteristic whoop appears; but it may be suspected when the child has been exposed to contagion, and when the coryza and redness of the fauces persist in spite of treatment.

In the diagnosis of bronchitis it is often more difficult to determine the primary cause than it is to distinguish bronchitis from other pulmonary affections. Yet the former is the more important; and it must be borne in mind that bronchitis is a frequent accompaniment of many febrile diseases, such as typhoid fever, measles, and whooping-cough; of diseases of the heart and kidneys, and of septic diseases and blood disorders. The primary will not be likely to be mistaken for the secondary disorder if one is upon his guard and insists upon finding a cause for each case that presents itself.

Measles can usually be diagnosticated from the first by the coryza, but

especially by the Koplik spots and the red spots upon the anterior half-arches of the soft palate, which appear usually several days before the eruption upon the body.

Bronchitis is a common and important early symptom of *typhoid fever*. The latter disease may be suspected when the fever, prostration, and headache are greater than they would be in bronchitis alone, and, especially if these symptoms coexist with a loose condition of the bowels, chilliness, and occasional nose-bleed.

Chronic Bronchitis.

Symptoms. This occurs most frequently in middle or later life. Its special feature is long duration, without fever and with comparatively little impairment of the general health. *Cough* is not constant; it is entirely absent for periods; it then returns, perhaps with increased severity, and lingers indefinitely.

Chronic bronchitis in its milder form consists in what is often called "winter cough." It attacks especially persons past middle life who have emphysema. It appears with the cold weather, and lasts until the following summer. The cough is not severe, though sometimes paroxysmal, and *expectoration* is scanty, non-purulent, and may be confined to the morning. *Dyspnœa* is not marked unless there is considerable emphysema. Acute exacerbations occur from time to time, and the tendency of the disease is to become worse from year to year, and to be more continuous, even persisting all summer.

In *dry catarrh*, or *catarrhe sec* of Laennec, paroxysms of cough occur on the slightest provocation, with the expectoration of small hard pellets, or without any expectoration. The patients with this form are emphysematous.

Physical Signs. These are those of bronchitis of the larger and middle-sized tubes. Large moist *râles* are more or less abundant, depending upon the degree of swelling of the mucous membrane, and the quantity and fluidity of the secretions. The *respiratory murmur* is roughened and less intense than normal.

W. Fox says that in chronic bronchitis there is commonly *hyper-resonance* from coexisting emphysema, but in acute exacerbations the bases may be dull from congestion or œdema. *Respiration* is harsh, and in some cases of senile bronchitis expiration may be both prolonged and high pitched when other signs of dilatation of bronchial tubes are absent. The *percussion-note* is clear.

The subjective symptoms of the patient consist, in ordinary cases, of a moderate amount of *dyspnœa* and *tightness across the chest*. At the onset of a fresh attack the symptoms may be those of acute bronchitis. The cough is paroxysmal, somewhat resembling that of whooping-cough, but without the characteristic whoop. It is usually severest on lying down and when rising in the morning.

The *sputa* of the severe forms of chronic bronchitis are usually copious and mucopurulent, the latter predominating. They vary in color from yellowish white to ashy, greenish, or black when the lungs are anthracotic

or collapsed. The quantity and character of the sputa vary more than in acute bronchitis. Sometimes they are very copious, consisting of serum mixed with mucus, constituting *bronchorrhœa*. More commonly they are scanty, glairy, and tenacious.

Diagnosis. This is made by noting the long duration of the disease without impairment of the general health, its relation to season, and the absence of physical signs of involvement of lung tissue.

Chronic bronchitis may be the result of repeated acute attacks, or, rarely, of only one. It is frequently found in association with gout, chronic heart disease, chronic endarteritis, Bright's disease, emphysema, asthma, and chronic alcoholism. It may alternate with other gouty affections, as articular inflammation or eczema, being relieved when the other manifestations are more marked. It also accompanies tuberculosis of the lungs. Climate and season have a marked influence; the disease is worse in damp, cold climates and in the winter months.

Chronic bronchitis can be diagnosticated from the cough of *aneurism* by the absence of the stridulous breathing, due to paralysis of one-half of the vocal cords, and by the local signs of a tumor of the vessel. Other tumors may cause cough by pressure, and the possibility of their existence should therefore be borne in mind.

Capillary Bronchitis, or Suffocative Catarrh.

This is bronchitis of the smaller tubes. It occurs most frequently as an extension of the catarrhal process from the larger tubes, but sometimes seems to attack the smaller tubes from the beginning, or coincidently with the larger tubes. Infants, young children, and the aged are most liable to it.

Symptoms. The disease begins with a succession of chills or chilliness, followed by high fever. The temperature may rise to 104° F. The skin is hot, the face flushed. The head and neck and the upper portion of the trunk may be covered with perspiration. The pulse rapidly increases in frequency.

The aspect of the patient from the first shows that the illness is graver than ordinary bronchitis. The face expresses anxiety, and in children the *alæ nasi* dilate in respiration, which is both accelerated and difficult (*dyspnœa*). The respirations may be as many as 80 to the minute, the pulse not being correspondingly rapid. *Dyspnœa* is more or less constant, but becomes urgent in paroxysms, and the patient may have to be propped up in bed to enable him to breathe (*orthopnœa*). It is *expiratory*: inspiration may be free and easy, or difficult; but expiration is always difficult and prolonged. In children the pause in the act of breathing takes place at the end of inspiration, instead of expiration.

Cough is more frequent and violent than in ordinary bronchitis, and the *expectoration* is viscid and difficult to raise. As the disease progresses, *dyspnœa* becomes more intense, and signs of insufficient aëration of the blood make their appearance (*cyanosis*). The lips and finger-nails become bluish, and the extremities cool and clammy. If the patient is unable to expel the tenacious secretions from his bronchial tubes, the further

progress of the case is that of rapidly developing cyanosis ; the breathing continues frequent, but is shallow and more labored. Children often have convulsions, followed by coma and death, while old persons sink into coma without preceding convulsions.

The *sputum* contains mucus, pus, occasionally blood-cells, granular matter, and sometimes fibrinous casts of the tubes. The micro-organisms found are *Micrococcus lanceolatus*, *Streptococcus pyogenes*, and *Staphylococcus aureus et albus*. Mixed infections are usually present.

Physical Signs (Plate XIX.). These are those of bronchitis of the larger and smaller tubes. Sibilant and sonorous *râles*, if present at first, give way to fine subcrepitant and crepitant *râles*, which speedily become moist and very abundant. As in ordinary bronchitis, the bases of the lungs posteriorly are the parts most involved. The *percussion-note* of both lungs remains clear, but there is apt to be *increased resistance*. The *fremitus* may be lessened in some areas, increased in others. If an area of *dulness* appears, it may be due to pneumonia or collapse of the lung ; if the former, there is usually an access of fever.

Plastic Bronchitis.

This is a form of bronchitis, usually chronic, the characteristic feature of which is the expectoration of fibrinous casts, which, when unravelled under water, are found to be solid casts of the smaller bronchial tubes. The casts are often tree-like in shape, showing that a bronchial tube and its smaller subdivisions have been occluded by the casts.

Persons of all ages are liable to plastic bronchitis, but it affects males about twice as often as females.

The subjective symptoms are *cough* and *dyspnœa* ; *hæmoptysis* occurs in about one-third of the cases. (Biermer.¹) The cough occurs in paroxysms, which are frequent and severe ; relief follows expectoration of the casts.

Hemorrhage may appear only as streaks of blood upon the casts, or may be considerable and follow their dislodgement. The *casts* themselves when ejected are usually coated with mucus, so that they appear as solid masses of sputum ; their arrangement into cylinders may not be suspected until they are agitated in water. The size of the cylinder varies from that of the little finger to that of a bodkin, but they do not often exceed the size of a goose-quill. The larger casts may be hollow, but the smaller ones are solid, and are arranged in layers. They are whitish or gray in color, and firm in consistence, but become softer as the disease improves. Microscopically, the casts are nearly structureless, consisting of a fibrillated base, with pus and mucous corpuscles, a few gland-cells, and, occasionally, blood-cells in the outer layers. Charcot-Leyden crystals and Curschmann's spirals are found.

The acute form is rare, and out of ten cases accepted by Biermer six proved fatal. The disease begins with *fever*, *dyspnœa* appears early, severe paroxysms of cough occur, sometimes hemorrhage. Death results

¹ Virchow, Handbuch der spec. Path. u. Ther., Bd. v., Abth. 1.

PLATE XIX.

FIG. 1.—Anterior Aspect.

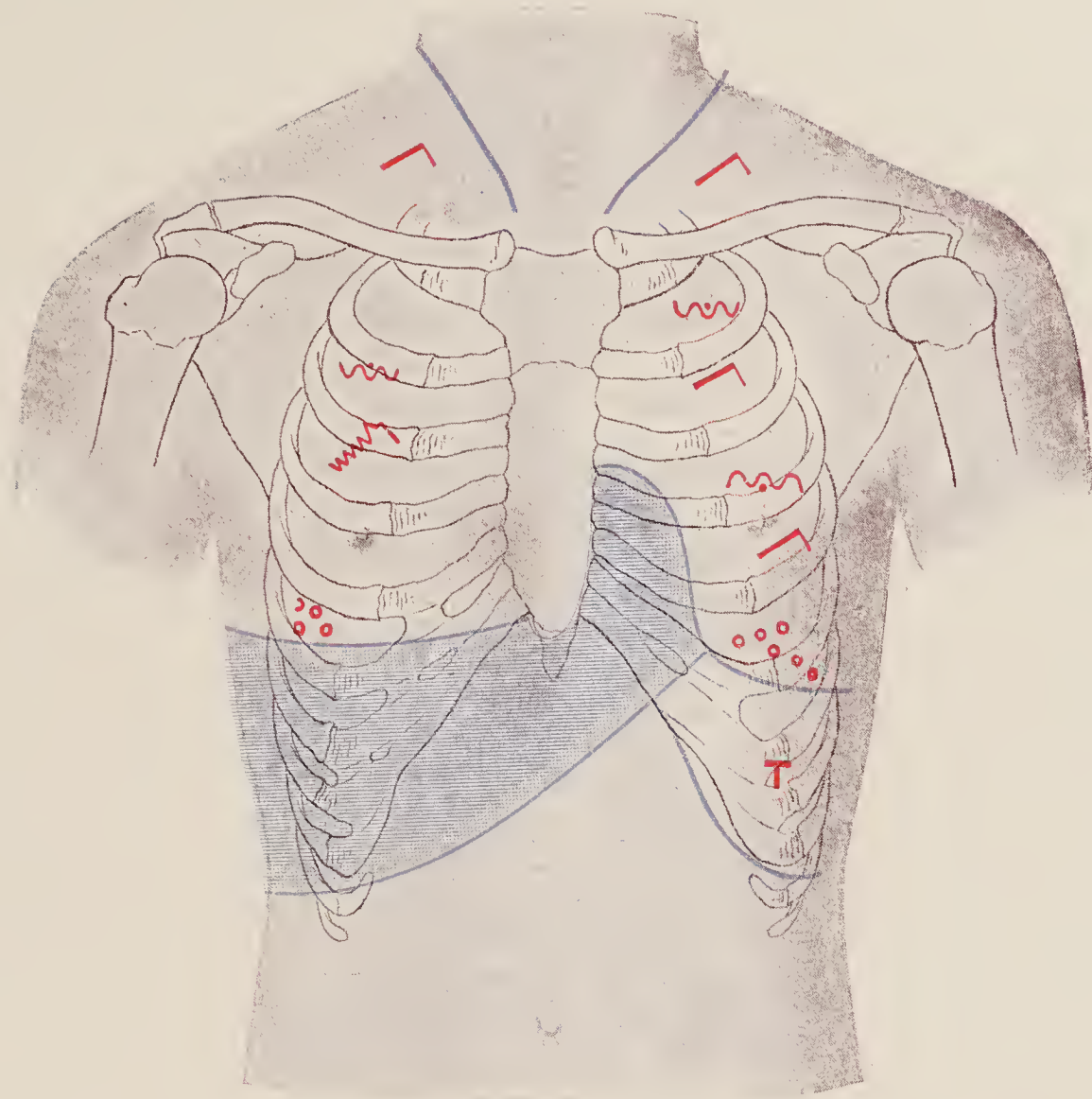
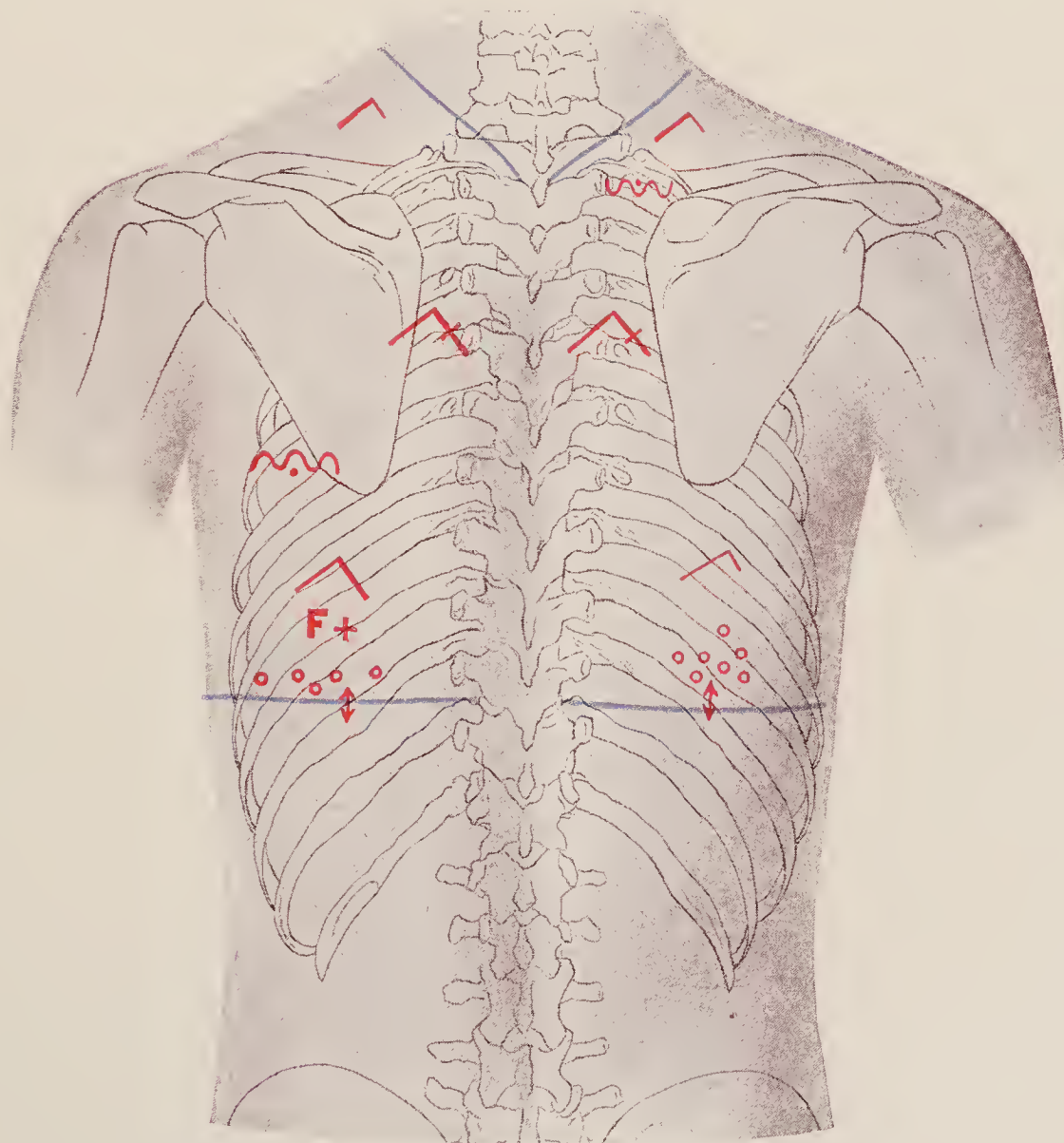


FIG. 2.—Posterior Aspect.



Capillary Bronchitis (early stage).

Rough or sharp breath sounds—expiration in places prolonged. Sonorous, sibilant and small moist rales. Local increase of fremitus.

from asphyxia. Grave symptoms are excessive dyspnoea, scanty expectoration, and drowsiness. Copious expectoration is a favorable sign.

Physical Signs. The casts obstruct the bronchial tubes. There is less air entering the part, hence there are *diminished fremitus* and *respiratory murmur* over the portions of lung supplied by the obstructed tubes. If collapse ensues, there is *dulness* on percussion; if the casts are dislodged, the murmur becomes normal or but slightly roughened. In unaffected portions of the lung resonance is clear or exaggerated, and the respiratory murmur remains unaltered.

Fuller says¹ that the upper portions of the lung are oftener affected than the lower portions.

Fetid or Putrid Bronchitis.

This name is applied to the condition in which the sputa have a highly offensive odor and are copious and semi-putrid. The odor is said by some to be due to microscopical sloughs, and by others to a special bacillus.

Putrid bronchitis may accompany (1) dilatation of the bronchial tubes; (2) chronic pneumonia; (3) phthisis or (4) empyema with a fistulous communication with a bronchus; or (5) it may occur independently.

Symptoms. The subjective symptoms are cough, irregular fever, and occasional chills. The *sputa* of fetid bronchitis have an odor of gangrene or feces. On standing they separate into three layers. The upper one consists of a greenish fluid or frothy layer; the second is sero-albuminous; and the third a thick granular deposit in which are masses the size of peas (Dittrich's plugs), and flakes consisting of granular detritus, and containing fatty crystals and bacteria, *Oidium albicans*, and crystals of leucin and tyrosin. (See Sputum.)

Physical Signs. These are those of chronic bronchitis, or of bronchitis and the conditions with which it may be associated (*q. v.*).

Diagnosis. This form of bronchitis is diagnosticated from *gangrene* by the absence of physical signs of disintegration of lung tissue and by the absence from the sputum of fragments of lung tissue and elastic fibres. Nevertheless, gangrene of the lung may be the final result of putrid bronchitis.

Infectious Bronchitis.

In addition to the bronchitis that attends the infectious disorders mentioned above, three forms are seen of an infectious nature which are properly classified among the infectious diseases. It is proper to refer to them now, as bronchitis is usually the most pronounced local manifestation. They are *influenza*, *whooping-cough*, and *hay fever*. The last only will be spoken of at present.

Hay Fever.

Hay fever is a specific catarrh of the respiratory passages, caused by the pollen of certain plants, principally the grasses. The attack begins

¹ Quoted by Peacock in Diseases of the Chest.

with *itching*, *burning*, and *lacrymation*, and *pain* in the *brow* or *eyeballs*. Subsequently there are *itching* or *pricking* of the *nasal mucous membrane*, frequent *sneezing*, and an irritating *watery discharge*. The mucous membrane of the nose is red and swollen. A similar condition obtains in the throat when it is affected. If the disease attacks the bronchial mucous membrane, a bronchitis is set up, which, if it differs at all from ordinary bronchitis, is more persistent and attended by greater dyspnoea, with asthmatic attacks.

Collapse of the Lung.

Collapse of the lung is a condition produced by exhaustion of air from the air-vesicles. It may affect alveoli here and there, or even a large section of the lung. Formerly such collapse was invariably looked upon as pneumonia, until Legendre and Bailly proved by forcible inflation that the air-vesicles had simply collapsed from absence of air. Collapse occurs most frequently in the course of bronchitis and in cases with feeble respiratory power. The bronchial twigs supplying certain air-vesicles, or tubes supplying sections of lung, become occluded to such a degree that no air can enter. The air already contained in the vesicles then becomes exhausted gradually until the vesicles are completely airless. The vesicles or sections of lung involved then return to the foetal condition. When the collapse is congenital, the term *atelectasis* is preferable. Anything which induces great muscular weakness predisposes to collapse of the lung; hence, in the aged and feeble, in wasting diseases, and in low febrile diseases of long standing, collapse is very apt to occur. But bronchitis is the most frequent and direct cause. The secretions which are poured out, and the swelling of the mucous membrane, occlude the tubes, and if the patient has not sufficient strength to expel the secretions, and by forced inspiration to expand the collapsing vesicles, collapse ensues.

Symptoms. Subjective symptoms are those of dyspnoea and insufficient oxygenation of the blood.

Physical Signs. If the symptoms are developed suddenly, and are accompanied by the appearance of dull areas in the lung without bronchial breathing, the diagnosis is tolerably certain; but when scattered lobules only are involved, the physical signs of collapse are absent, and its existence must be a matter of inference.

In life the diagnosis of the condition consequently is difficult. The site of collapse, being airless, is, of course, *dull on percussion*. The *respiratory murmur* is more likely to be *faint* or *absent* than to be increased in intensity or approach the bronchial. Nevertheless there is sometimes heard a faint bronchovesicular expiration.

When œdema is superadded to collapse, moist crepitant *râles* are heard, difficult if not impossible to distinguish from those of pneumonia. Respiration is embarrassed, and is accompanied by sucking-in of the lower part of the chest in inspiration. Sometimes the plug of mucus which occludes the tubes becomes dislodged while the physician is auscultating, and then the respiratory murmur will be heard, accompanied by a suc-

cession of crepitant râles, which disappear after a few inspirations. The dull areas, as a rule, are less persistent than those of pneumonia; thus it may be found at successive examinations that one area has cleared up and another has become dull. Stress is laid by some writers upon the signs of emphysema surrounding collapsed areas. But this does not give assistance in the cases in which most help is required—cases in which there is diffuse bronchitis with more or less œdema.

Diagnosis. From *lobar pneumonia* the diagnosis is easily made by the difference in the physical signs, and by the absence in pulmonary collapse of inflammatory symptoms, by the lower temperature, and the difference in onset.

The diagnosis from *bronchopneumonia*, or *catarrhal pneumonia*, is beset with greater difficulties. But here also the low temperature, and the fact that the physical signs and the location of the dull areas are subject to rapid changes, are of aid in diagnosis.

Bronchopneumonia, or Catarrhal Pneumonia.

This is a pneumonia occurring secondarily to bronchitis, and characterized by the development of areas of consolidation in both lungs and the persistence of a bronchitis of the middle-sized or smaller tubes. In proportion as the areas of consolidation are large, the symptoms and physical signs approach those of lobar pneumonia. It is more common in children and in debilitated persons. It is the chief form in infants. It is frequently *secondary* to measles, diphtheria, scarlet fever, and pertussis.

As *aspiration pneumonia*, it occurs when food, septic particles, blood, or tissue enter the lungs during the loss of sensibility of the larynx in apoplectic, uræmic, or other forms of coma, and in operations about the upper air-passages and mouth. It is a fatal complication of tracheotomy. It is frequently of *tuberculous* origin.

Symptoms. Catarrhal pneumonia, except the aspiration form, develops gradually, and it may not always be easy to mark the point at which the bronchitis which precedes merges into pneumonia; but as a rule there is more or less *chilliness* (rarely a decided chill) and an access of *fever*. There is usually greater *prostration* than in the lobar form, in proportion to the amount of pneumonia present. The *pulse* is more *frequent* and more likely to be *feeble*. *Cough* and *expectoration* are marked symptoms. The *sputum* is tenacious and glairy, not rusty. It contains streptococci and staphylococci in much greater numbers than are found in ordinary bronchitis; fatty epithelial cells, epithelium, fat-globules, and diplococci.

Dyspnœa is more extreme than in lobar pneumonia. The respirations are excessively rapid—60 to 80 per minute; *cyanosis* rapidly ensues. The finger-tips become blue, the face dusky. The fever does not rise as high as in the lobar form. At first the skin is hot and dry; later it becomes cold and clammy, and in the tuberculous form sweats are common. The duration of the disease is usually much longer than in lobar pneumonia.

In the common form seen in infants the symptoms of *asphyxia* set in at variable periods in the course of the disease. General cyanosis super-

venes. Stupor sets in, the hurried respirations grow shorter and more gasping, the pulse becomes excessively rapid and feeble, the extremities cool and clammy; with the stupor the cough abates and the breathing becomes more shallow. The lungs fill with fluid mucus, and the child drowns in its own secretions, or cardiac paralysis sets in after dilatation of the right heart.

Examination of the *sputum* shows an abundance of the streptococci and staphylococci and the special micro-organism which belongs to the primary infection, as that of influenza, diphtheria, or tuberculosis.

Physical Signs. (Plate XX.) These are those of bronchitis, with here and there larger or smaller areas of *consolidation*, over which the râles are finer and closer set; the *percussion-note* is *dull*, and the *respiratory murmur* is *bronchial* or *bronchovesicular*. An entire lobe may be consolidated. Areas of collapse and portions more or less œdematous combine to make the more complex physical signs. While both lungs are affected, they are not usually so to the same extent. It is said that the apices are more prone to involvement in this than in the lobar form; and some writers (Osler) look upon it as almost, if not always, of tuberculous origin.

Diagnosis. The affection is distinguished by (1) its pathological antecedents and causal relations; (2) its gradual onset; (3) its distribution in both lungs; (4) the preponderance of physical signs of bronchitis over those of consolidation; (5) the extreme dyspnœa and cyanosis with a lower temperature than in lobar pneumonia; (6) the onset of carbon dioxide poisoning; (7) the long duration and gradual decline. The *tuberculous* form is distinguished by (1) the history of exposure to infection or of a focus of infection in the body, glands, or joints; (2) the longer course; (3) the delayed asphyxia; (4) the rapid emaciation; (5) the diffused sweats; (6) the physical signs of consolidation and subsequently of cavity at the apex; and absolutely by (7) tubercle bacilli in the expectoration, coughed up or vomited. I have seen a child aged fifteen months, of a tuberculous mother, completely recover. The tuberculous form is common in colored infants.

Lobar Pneumonia, or Croupous Pneumonia.

For consideration of this disease, the reader is referred to the section on Infectious Diseases, Chapter I., Part II. (Plate XXI.)

Chronic Interstitial Pneumonia.

Cirrhosis, fibroid phthisis, and chronic interstitial pneumonia are names given to a condition of chronic induration of the lung, caused by interstitial over-growth of fibrous tissue. Obliteration of the air-vesicles and contraction of the lung result from the over-growth. The bronchi are frequently dilated, and cavities and gangrene may occur. The disease is rare except as the result of tuberculosis, but it may follow pneumonia and pleurisy, and it is said to be caused by inhalation of fine particles of steel

PLATE XX.

FIG. 1.

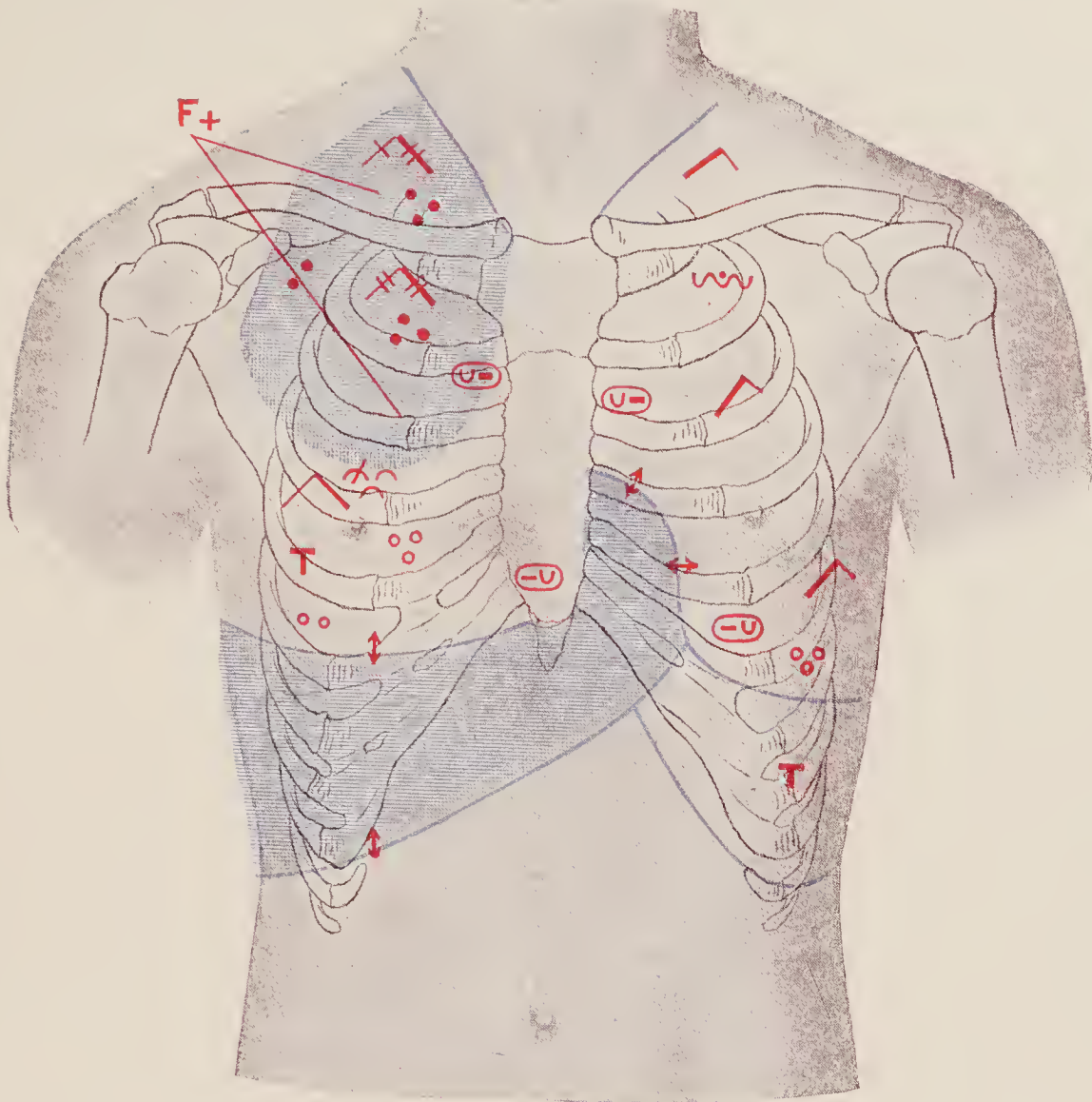
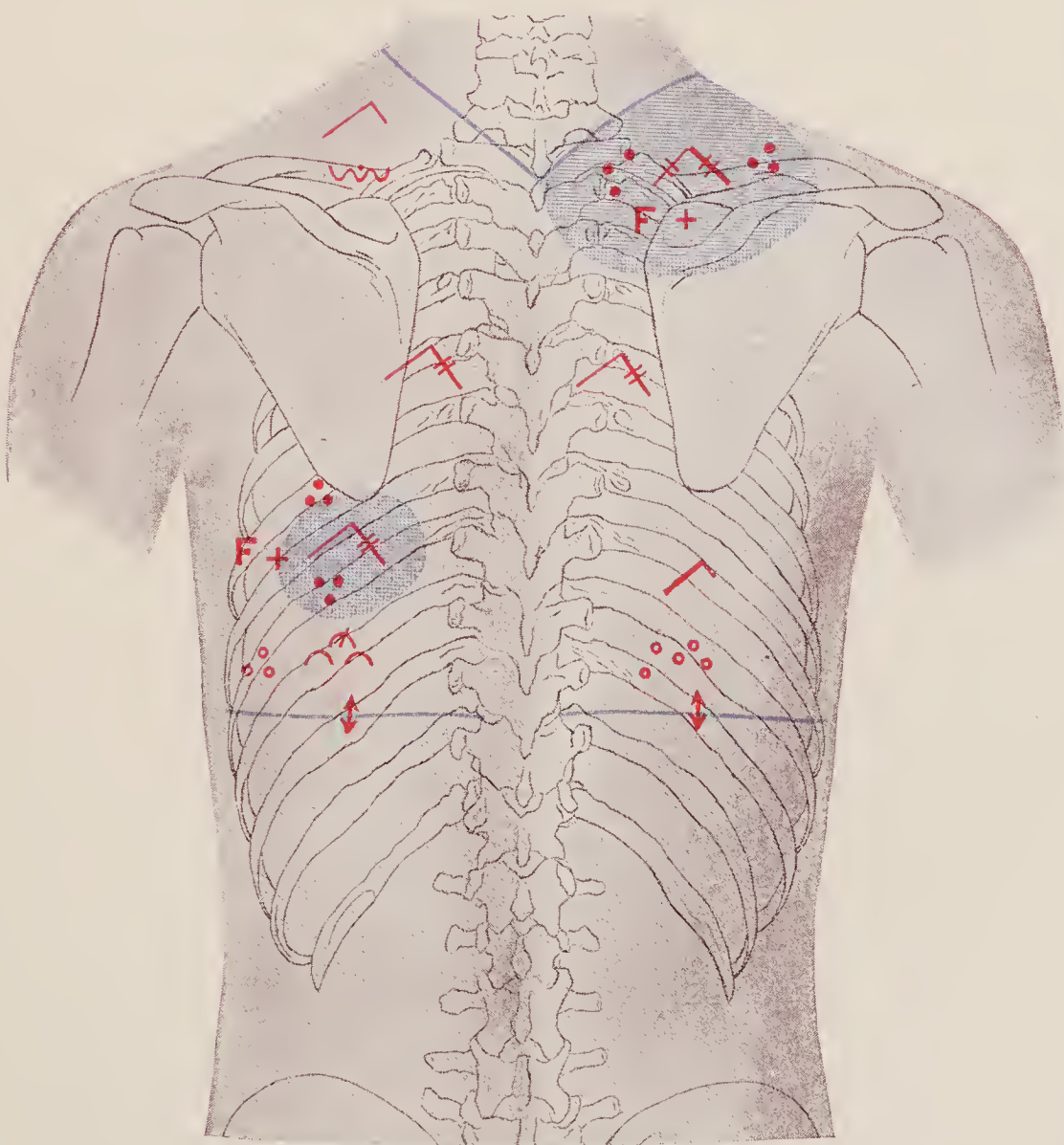


FIG. 2.



Bronchopneumonia.

Consolidation in the right upper and the left lower lobes. Physical signs of bronchitis over both lungs.

PLATE XXI.

FIG. 1.

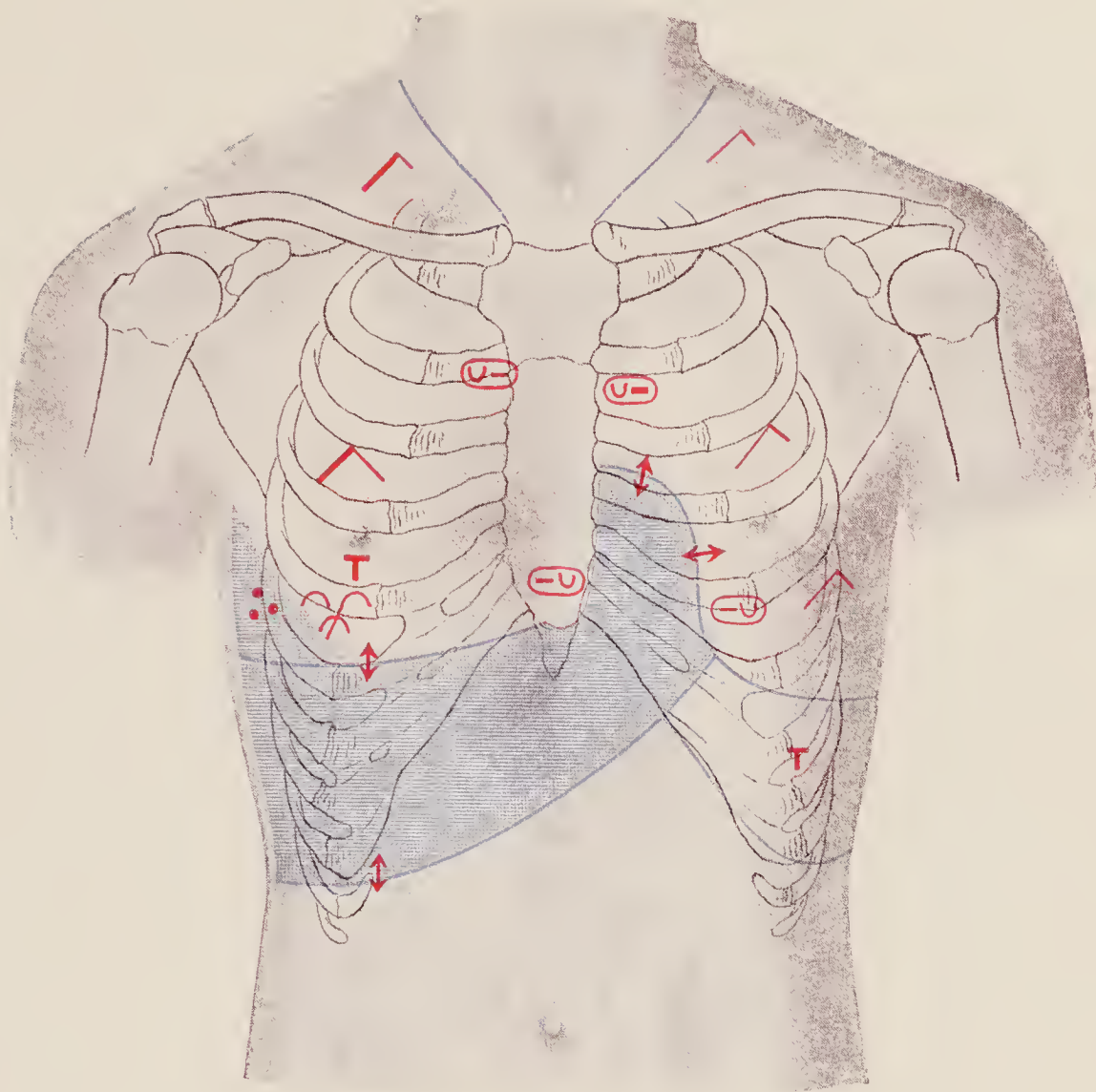
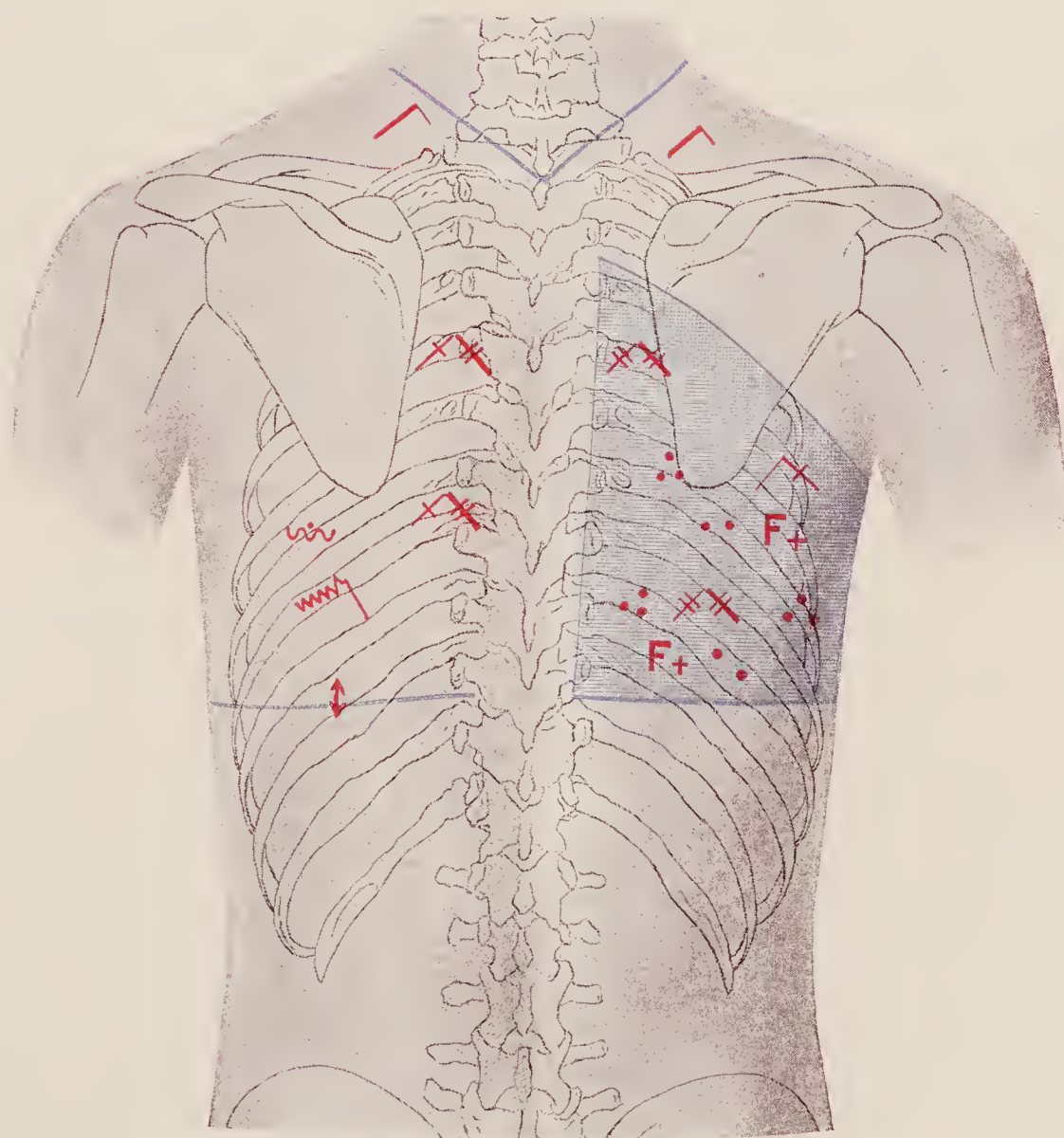


FIG. 2.



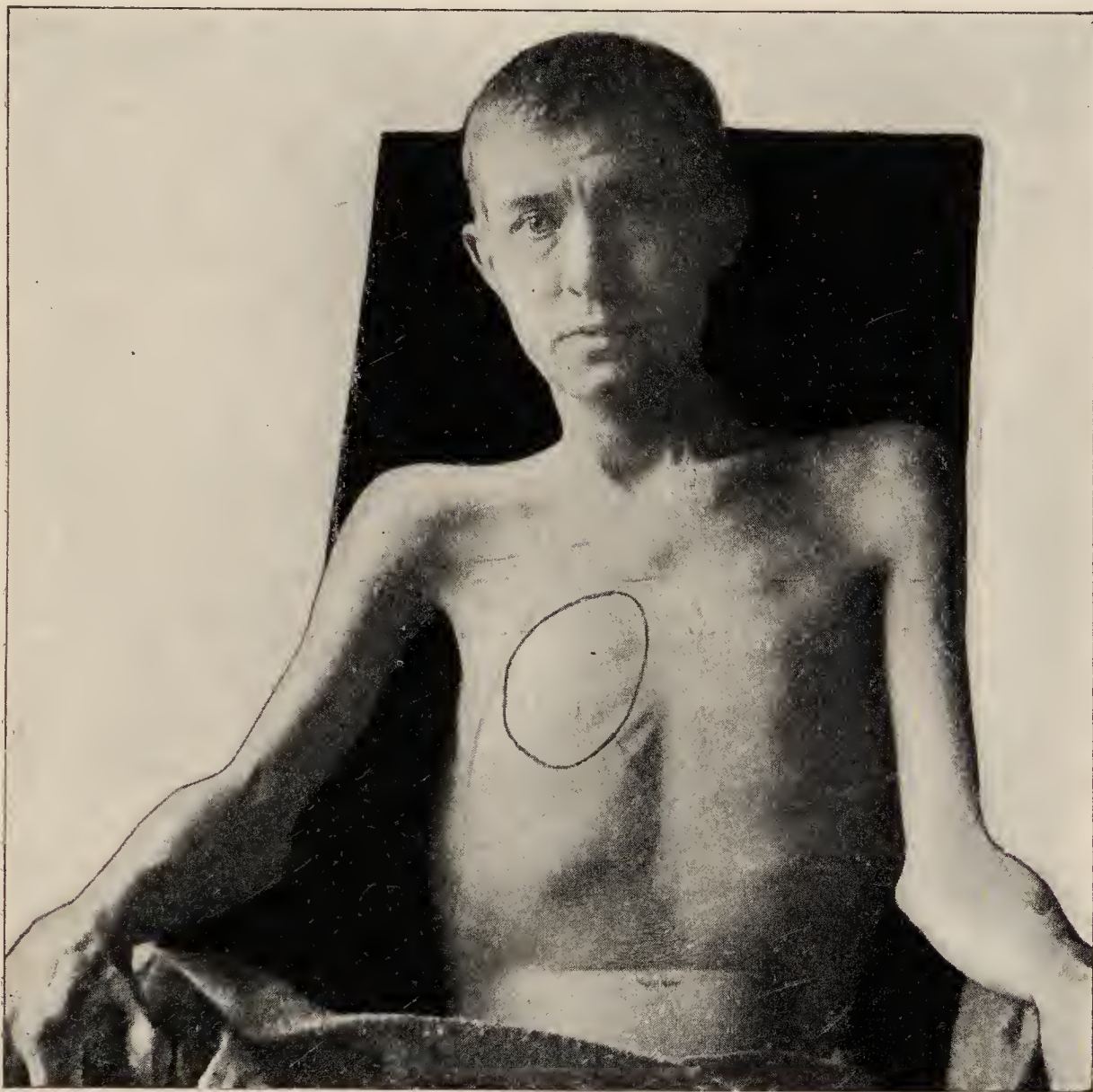
Lobar Pneumonia.

Consolidation of the right lower lobe. Transmitted bronchial breathing and signs of bronchitis over the left lung posteriorly.

or cotton. *Pneumonokoniosis* is the term, first employed by Zenker, for the chronic interstitial pneumonia due to the inhalation of dust.

Symptoms. The disease runs a very chronic course, attended by *cough*, and mucopurulent and sometimes bloody *expectoration*, even *hemorrhage*; but there is no fever and not much loss of flesh. *Dyspnœa* occurs on ascending heights only. Dilatation of the right heart is likely to ensue, with cardiac murmurs and increased lateral dulness and increase of dyspnœa. Death is hastened by the disease, and is often brought on by acute pneumonia.

FIG. 344.



Fibroid (tuberculous) phthisis; right apex. Heart displaced as indicated by oval. (Original.)

Physical Signs. (See Plates, Bronchiectasis.) **Inspection.** The disease is *unilateral*. The chest-wall is *retracted*. The ribs are drawn together, so that the interspaces are obliterated. The shoulder is drawn over the sunken thorax. The *spinal column* is *curved*. The *heart* is *displaced*; it is drawn toward the affected side. If the right lung is the seat of disease, an impulse is seen to the right of the sternum; if the left, the præcordial area of impulse is increased and extends upward. There is *no expansion* whatever (immobility) of the affected apex or base. The healthy lung is the seat of compensatory emphysema. (See Fig. 344.)

Palpation. Inspection is confirmed. *Fremitus* is *increased*, especially at the apex. At the base, pleural thickening lessens the fremitus.

Percussion. The physical signs show *increased density* of lung tissue, with *dulness* on percussion, or, over a dilated bronchus, a tympanitic or amphoric note.

Auscultation. The *respiratory murmur* is *bronchial*, or, over a dilated bronchus, has a hollow sound. At the base breath-sounds are feeble, distant, or absent. *Râles* are also heard.

Pneumonokoniosis (also known as *anthracosis*, coal-miners' disease; *siderosis*, from metallic dust; *chalicosis*, from mineral dust, as in stone-cutters' phthisis). In this form there is a history of *exposure* to the irritating particles for a considerable period, during which time *cough* develops, gradually increases, and the *general health fails*. *Emphysema* simultaneously arises, causing *dyspnœa*. The patients wheeze, cough in paroxysms, and expectorate *sputum* which contains the dust-particles. In anthracosis it is black. On microscopical examination the special dust-particles are often found. The symptoms of emphysema and chronic bronchitis predominate. Tuberculous infection may take place late in the disease.

Pulmonary Tuberculosis.

For convenience of diagnosis the specific inflammation of the lungs caused by *Bacillus tuberculosis* will be considered in this section. If a strict ætiological classification were followed, it would be considered among the infectious diseases.

Clinically, we see tuberculosis in the lungs manifesting itself in one of the forms of acute pneumonic phthisis, acute miliary tuberculosis, and chronic ulcerative phthisis. (See Chapter I., Part II.)

Tuberculosis of the lungs, pulmonary phthisis, and consumption are names applied to an infectious and mildly contagious disease of the lungs, caused by the tubercle bacillus, appearing in an acute and chronic form, and characterized by cough, fever, sweats, more or less rapid emaciation, purulent expectoration containing elastic fibres and tubercle bacilli, and by peculiar physical signs.

Acute Pulmonary Tuberculosis, Acute Phthisis, Acute Pneumonic Phthisis, Galloping Consumption. This disease may be primary or may be secondary to a localized area in the lung, causing rapid infection, or to tuberculous pleurisy, tuberculous peritonitis, or to tuberculosis of some other organ.

Symptoms. Its onset is usually marked by *cough*, *fever* with or without chills, *dyspnœa*, and sometimes *hæmoptysis*. The fever rises to 103° or 104° F., and is of a continued type (lobar pneumonic form), or rapidly assumes a hectic type, accompanied by restlessness and exhausting night-sweats, anorexia, and rapid emaciation. *Prostration* is extreme, but the mind is at first clear and the spirits cheerful. *Cough* increases; the *expectoration*, at first mucoid and scanty, but often tinged with blood, becomes more copious and mucopurulent. The *bowels* may be loosened or constipated. The *urine* may show the diazo reaction.

When death takes place without more decided pulmonary symptoms, the tuberculosis has been secondary to tuberculosis elsewhere, or death is the result of a general miliary tuberculosis.

PLATE XXII.

FIG. 1.—Anterior Aspect.

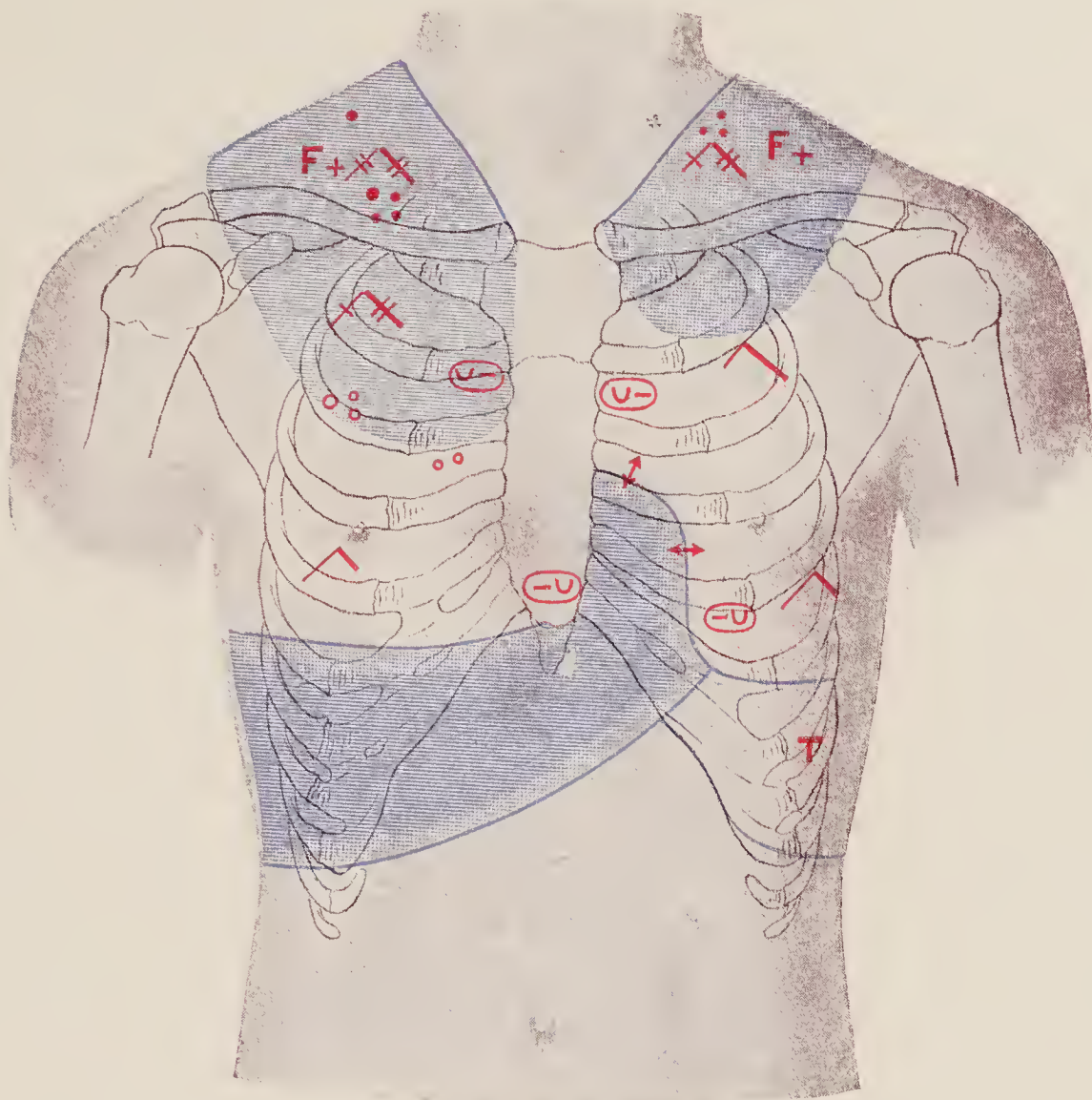
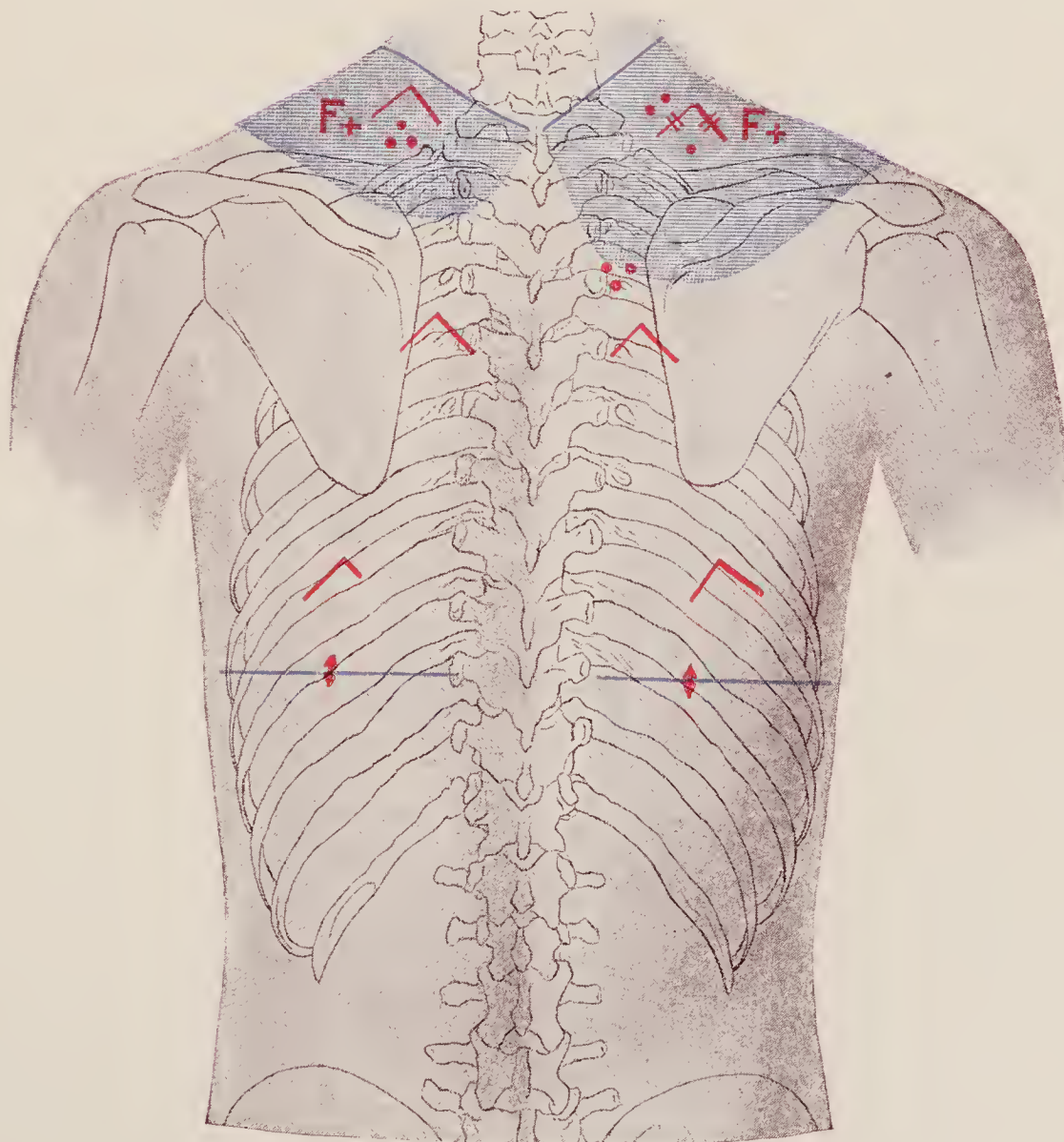


FIG. 2.—Posterior Aspect.



Acute Pulmonary Tuberculosis.

Consolidation of the entire right upper lobe and of the left apex.

When the acute pulmonary tuberculosis is primary, the character of the disease is soon made clear by the early development of consolidation of the lungs, usually of an apex first, rapidly followed by softening and the formation of cavities. The *sputum* becomes mucopurulent, is frequently streaked with blood, and pure blood is often coughed up. The sputum contains yellow elastic tissue and abundant tubercle bacilli. The patient often presents a *cachectic* appearance; emaciation has been very rapid, and has reached an extreme degree; there is frequently a red flush about the cheek-bones, which, with the bright eyes, contrasts strongly with the hollow cheeks and temples, and the white, wasted hands and clubbed fingers with bluish nails. *Cyanosis* is shown in the dusky countenance and blue finger-tips. The *exhaustion* becomes extreme. The *sweats* are profuse. The appetite is lost. *Diarrhœa* may set in. *Remissions* may take place, even in acute cases; for a time the fever and more aggravated pulmonary symptoms are in abeyance. The typhoid state ensues in some cases. *Death* takes place from exhaustion and heart-clot or from meningeal tuberculosis. The *duration* is from two to six weeks.

The patient's *mental attitude* is often peculiarly and characteristically hopeful. He expresses himself as better each day, though he is occasionally subject to despondency, and is sure that if he could only gain a little strength he would soon be well.

Sometimes, especially in children, the disease is *latent*. The patient suffers from weariness, the cheeks flush easily, the pulse is readily disturbed, there are nocturnal fever and occasional sweats. Emaciation proceeds very gradually, and a long time may elapse before disease is demonstrable.

In a few cases the *cerebral symptoms* are so pronounced as to mask the pulmonary, and in other cases there is actual coincident involvement of the cerebral meninges.

Physical Signs. (Plate XXII.) These are those of consolidation, often without conjoint pleurisy. The apex is usually first invaded. There are *diminished movement*, *increased fremitus*, and *dulness on percussion*. At first the *breathing* is *bronchovesicular*. It rapidly becomes *bronchial*. At first small moist *râles* are detected. Later they become large and gurgling. A *pleural friction*-sound may be heard. It may be heard first above the spine of the scapula behind, above the clavicle in front, or high up in the axilla. The upper lobe of the right lung may be affected at first, or the anterior portion of the middle lobe. The physical signs may be observed first in the axillary region of either side. The consolidation extends to the remainder of the lung, being preceded by physical signs indicating gradual encroachment upon the air-containing structure. The respiratory murmur is harsh, but soon becomes bronchovesicular and then bronchial (lobar pneumonic form). As consolidation progresses in the middle and lower portions of the affected lung, signs of cavity or multiple cavities appear in the upper. (The whole of a lobe may be the seat of small cavities filled with mucopurulent or purulent fluid.) *Cavernous breathing* and *pectoriloquy*, or the *bronchial sniff* of consolidation, become more pronounced. The dull note of consolida-

tion is relieved by a *dull tympanitic* or full tympanitic note. Now moist râles of all degrees are heard (*bronchopneumonic form*). Above they are gurgling; below, small and large moist râles. If the progress is not too rapid throughout the lung first affected, signs of invasion are found in the remaining lung, usually at a point corresponding to the primary focus in the original lung. The apex therefore is first invaded in most cases. Infection of the second may begin earlier than the signs in the first lung would lead one to anticipate. The rapid invasion of one lung compels compensatory emphysema of the other. The increased movement, with harsh or puerile breathing, without change in fremitus or in pitch and tone on percussion, masks many small consolidations.

Diagnosis. In the earliest stages, before the invasion of new territory is pronounced, the cases are involved in doubt. Pulmonary tuberculosis may be confounded with pneumonia until the sputum is secured and bacilli are found.

In *pneumonia* we have the pronounced rigor, the rapid rise of temperature, the altered pulse-respiration ratio, the hot, dry skin, the sticky, viscid sputum, containing the pneumococcus, the peculiar changes in the urine, leucocytosis, the occurrence of herpes, and the termination by crisis, to point to the nature of the process. Emaciation is not marked; there are no such profuse sweats as the repeated drenchings we see in pneumonic phthisis; anæmia is not so pronounced. Then cavity formation does not take place, or at least occurs rarely. In pneumonia the fever is of a continued type; in phthisis it is often intermittent or remittent. The sputum is more purulent in acute pneumonic phthisis. Finally, the history of exposure to infection, the primary occurrence of tuberculosis elsewhere, the secondary occurrence of tuberculosis in other organs after the lung-invasion, the longer duration—aid in determining the true affection. Inoculation of animals may be resorted to in doubtful cases.

Acute Miliary Tuberculosis of the Lungs. Symptoms. These are high fever, rapid emaciation, hurried breathing, rapid pulse, duskiness of face and extremities, more or less stupor, delirium, and the development of the typhoid state, with prostration and the occurrence of profuse sweats. Intestinal symptoms, as flatulence and distention, may be pronounced, and diarrhœa may form a prominent feature.

The onset is abrupt or may follow a period of malaise. In some instances the tuberculous process is more advanced in some situations than in others, giving rise to special local symptoms. Thus, recently, a patient was admitted to the Presbyterian Hospital with stupor and moderate delirium. He had fever, rapid pulse and breathing, and a peculiar dry, harsh skin. There were albuminuria, casts and blood in the urine, and it was thought he had uræmia. The temperature range was irregularly intermittent. The diagnosis was established later because of the development of undoubted secondary tuberculosis in other organs. At the autopsy general tuberculosis was found, with primary tuberculous ulceration in the bladder, the ureters, and renal pelves.

Physical Signs. These are negative or are those of bronchitis. There is resonance or hyper-resonance on percussion. The latter is not uncommon.

Diagnosis. Hurried breathing and cyanosis are distinctive features, out of all proportion to the physical signs, and, on this account, of diagnostic significance. Acute miliary tuberculosis must be distinguished from typhoid fever, septicæmia or pyæmia, and malignant endocarditis. It is distinguished from *typhoid fever* by the absence of successive stages in the course of the disease; in typhoid fever the evolution of the disease is more characteristic than its symptoms. The headache of the first week, finally disappearing, is noteworthy. The special range of temperature, the onset, the fastigium, and the defervescence at definite periods in the evolution of the disease, are of diagnostic value. Cyanosis is more constant and marked in tuberculosis. The skin and capillaries have more tone in typhoid fever than in tuberculosis, at least in the first two weeks. Hyperæmia follows irritation in typhoid; pallor, with duskiess, in tuberculosis. The eruption, with its specific mode of development, belongs to typhoid fever alone. The stools, the enlarged spleen, the vascular tone are suggestive of typhoid fever. The spleen enlarges earlier in the disease in typhoid fever. Bacteriological examination may be of service. The occurrence of intestinal hemorrhage, pointing as it does to typhoid fever, is a welcome sign in cases in which the diagnosis is obscure. I have never seen it in tuberculosis. In typhoid fever the reflexes (knee-jerk) are never absent; in tuberculosis, if the meninges are involved, they are variable, present one day, absent the next. The Widal test is important. The diazo reaction in typhoid fever is of some service, although it also occurs in tuberculosis. (See Urine.) It does not come on until later than the fifth day in typhoid. It disappears at a certain time in the involution of typhoid; it continues indefinitely in tuberculosis. (See Chapter I., Part II.)

The distinction of tuberculosis from *septicæmia* or *pyæmia* and *malignant endocarditis* is often difficult. We must search for local areas of septic or pyæmic infection. The ears, the teeth, the bones, the veins, the heart, the pelvic organs in females, the rectum, the genito-urinary tract, all must be carefully examined. Hemorrhagic infarcts, or metastatic abscesses, may be found which point to the original conditions. The eye-ground may show hemorrhages. The skin and mucous membranes may exhibit minute capillary hemorrhages or infarcts. They are the size of a pin-head, do not disappear on pressure, and are not elevated. The spleen is more likely to be enlarged in the septic affections. The respirations are not so rapid as in tuberculosis. Cyanosis is a distinctive feature of tuberculosis. The physical signs of endocarditis may be determined, and subsequently embolism or thrombosis prove the nature of the process.

Chronic Tuberculosis, Chronic Ulcerative Phthisis. Chronic tuberculosis or phthisis is much more common than acute tuberculosis, from which it is distinguished by its slow progress and by periods of remission, during which the disease may be arrested temporarily or permanently.

Symptoms. The disease may begin in a variety of ways. The most common mode of origin is in an ordinary *bronchitis* with which pleurisy is occasionally associated. Previous to this the patient may have been in good health, but generally the health has been impaired for some time.

The bronchitis may be simple or part of influenza, measles, whooping-cough, or some other specific disease.

The bronchitis usually proves obstinate, and by and by the patient will be found to have *lost strength*, and usually some *weight*. There is often a slight evening *rise of temperature*, and occasionally *nocturnal perspirations*. The appetite is impaired, and *anorexia* may exist. *Cough* is rarely absent, especially during the night or on waking in the morning; it may, however, be so slight as apparently to have escaped the notice of the patient. When characteristic, it is dry and hacking. *Expectoration* is scanty and mucoid, but occasionally it may be tinged with blood. It should be remembered that children and old persons sometimes do not expectorate, and that, as a rule, women are more inclined to suppress expectoration than men. No tubercle bacilli may be found in the sputum after repeated examination; but if examinations are continued, they will appear sooner or later.

Instead of developing after a bronchitis, as we have just described, it may set in suddenly under the guise of a pneumonia, more frequently of the catarrhal form. The symptoms and physical signs do not differ essentially from those of pneumonia, except that the expectoration is more likely to be profuse, mucopurulent, and blood-streaked, and bacilli are found in it; the fever is more hectic in type, and night-sweats are common. After the patient convalesces from such an attack he continues weak, does not gain flesh readily, still has a cough with expectoration, and evening fever with occasional night-sweats.

In some cases fever, emaciation, and weakness progress for some time before pulmonary symptoms arise.

In still other cases the invasion of the disease is by sudden *hæmoptysis*, which is oftener copious than not. Several such hemorrhages may occur in rapid succession, or there may be only one. Moreover, its disappearance may not be followed, or at least not immediately, by any further pulmonary symptoms; more commonly, however, it is followed by fever, cough, and expectoration.

In still other, but rarer cases, the pulmonary disease is latent, being marked by *gastric* or *peritoneal symptoms*, or by a general *anæmia*.

By whatever path invasion comes, the physician should be on the lookout for it, especially in a young adult predisposed by heredity or environment to tuberculosis. The recognition of the disease in its early stage requires the greatest skill, which in turn is recompensed with the highest reward, since the disease is then curable.

The further progress of a case of tuberculosis of the lungs, after consolidation has once become manifest, is very variable. It may be arrested at this point permanently, cure resulting from cicatrization. More frequently there is temporary arrest of the process; fever lessens or ceases entirely, the pulse resumes its normal rate, appetite improves, and there is a gain in flesh and strength. Cough and expectoration are more likely to persist than the other symptoms, but with the other improvement they diminish in frequency and copiousness.

After a longer or shorter time, as the result of reinfection from the old focus excited by acute bronchitis or by some depressing influence, the

PLATE XXIII.

FIG. 1.—Anterior Aspect.

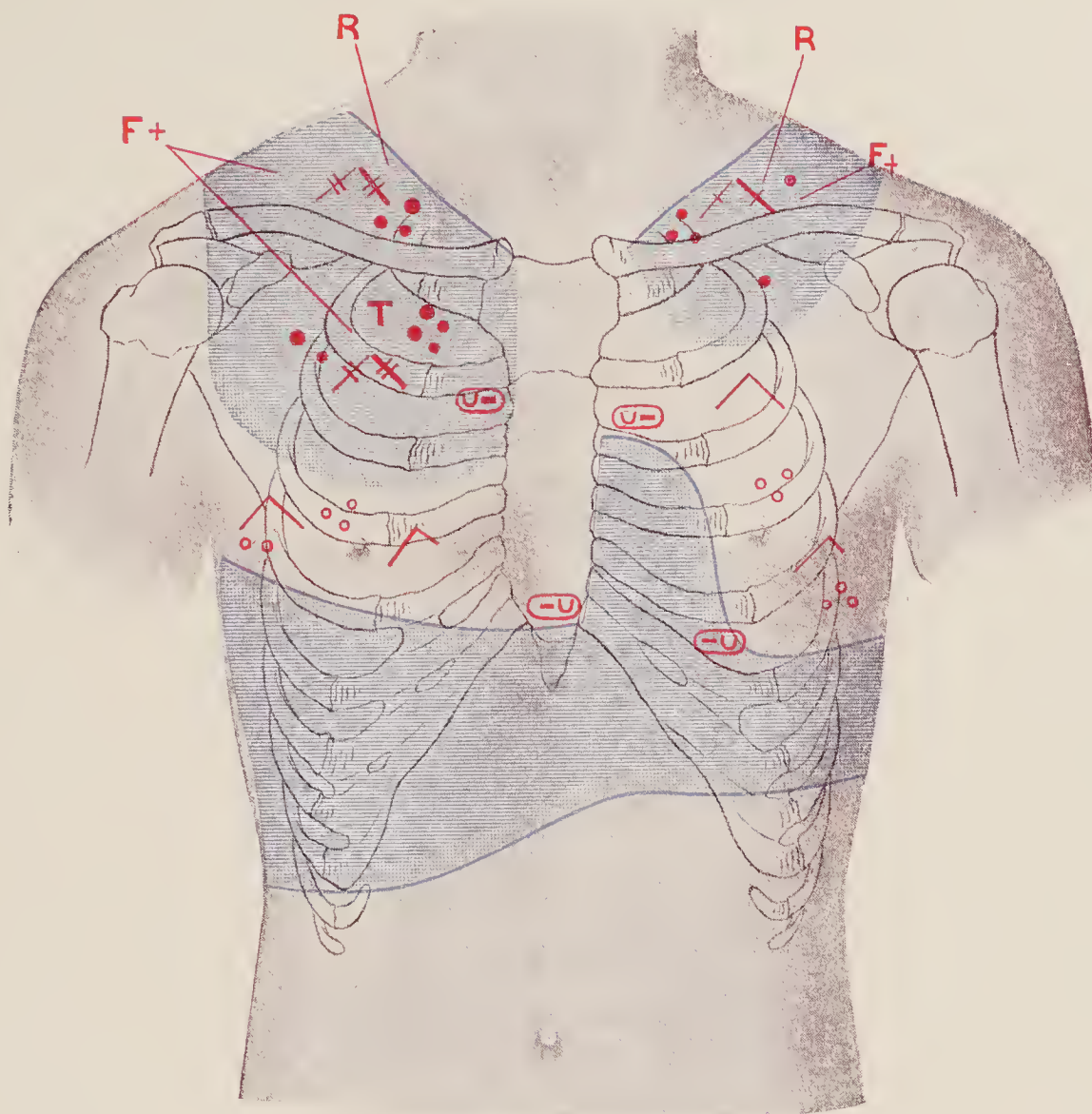
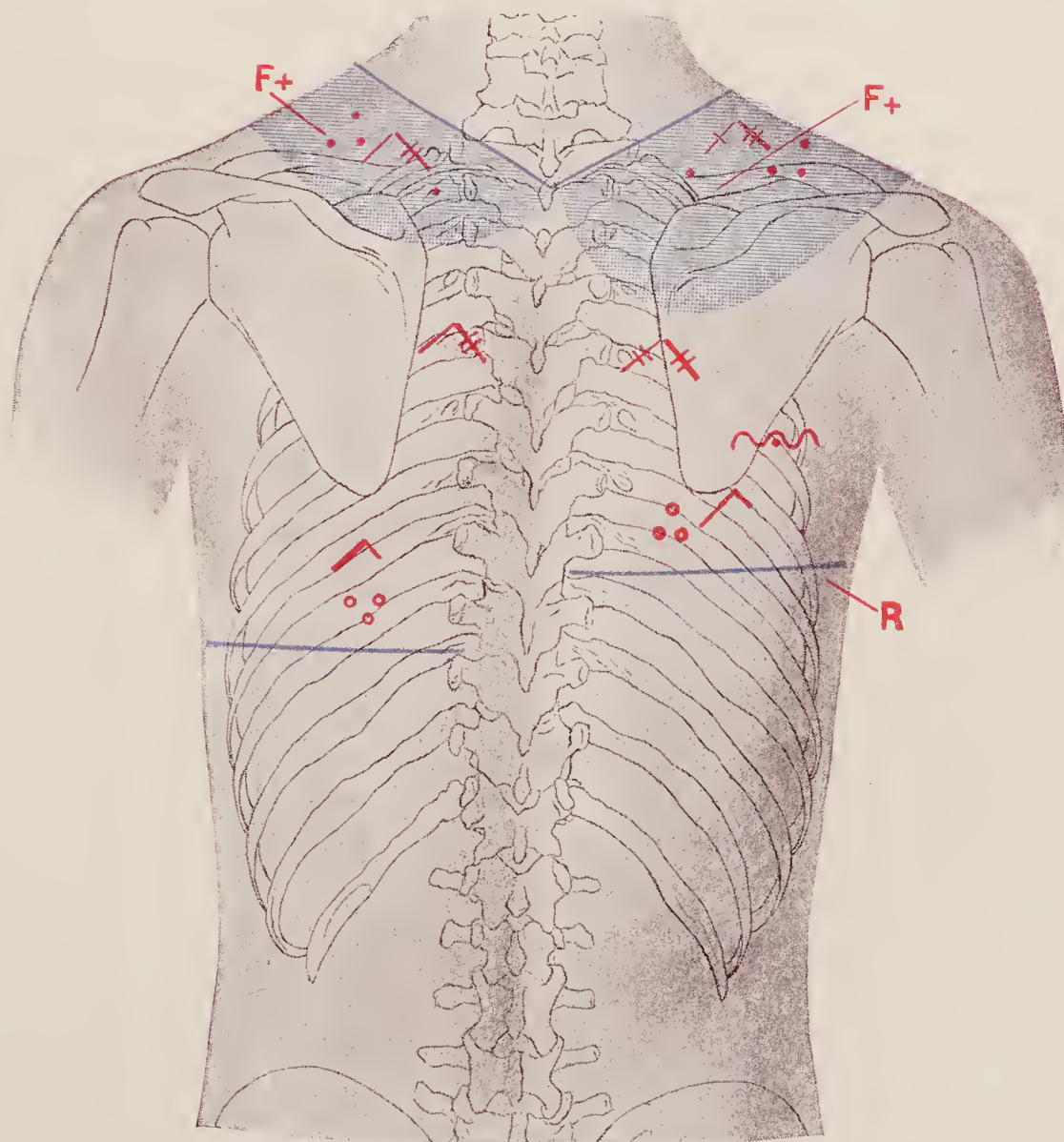


FIG. 2.—Posterior Aspect.



Chronic Pulmonary Tuberculosis.

Consolidation with cavity formation. Chronic pleurisy with loss of respiratory movement of lung margins. Retraction.

tuberculosis is relighted, so to speak, and runs much the same course, the lung being left more diseased and the general health worse after every such attack. Nevertheless, there may be long intervals between such attacks, the patient in the meantime continuing in fair health. Thus the disease may linger or recur for years, the patient not ill enough to be confined to the house, and not well enough to withstand hard work or great exposure. Slowly, by ulceration and suppuration, the lung tissue is wasted and cavities are formed. Before there are large cavities at an apex the base of the same lung becomes consolidated by the production of tuberculous material, and before one lung is extensively diseased the apex of the opposite lung is attacked, the process being repeated in it if the patient lives long enough. Instead of reinfection from an old focus, new infection may take place, giving rise to the old train of symptoms, or setting up some acute disease. During this time the patient is liable to an attack of acute pneumonia, pleurisy, bronchitis, or general miliary tuberculosis. He is also liable to sudden death by hemorrhage. In a number of cases the intestines and peritoneum become affected, and abdominal pain and diarrhoea are superadded as symptoms.

As a rule, the patient *gradually sinks*. The later stages are marked by increasing cough and dyspnoea, which are very distressing and prevent sleep. Expectoration is more copious, purulent, and is raised with increasing difficulty.

The appetite is poor and capricious, or anorexia is complete. The heart becomes more and more feeble, the fever is *hectic* and accompanied by *exhausting night-sweats*, the feet and limbs swell, and acute cramp-like pains are felt in the legs, probably caused by thrombosis of the veins.

Emaciation is extreme, scarcely anything but skin and bone being left. *Death* occurs from perforation of an intestinal or gastric ulcer, from hemorrhage, or more commonly from exhaustion, and from asphyxia caused by oedema of the lungs.

Physical Signs. (Plate XXIII.) These depend upon the lesions. It is often possible to detect all stages of the tuberculous process, from early consolidation to large cavity, in the same patient. When the disease begins as an obstinate bronchitis, there is found at the apex of the lung a small area over which, on percussion, there is *increased resistance*, with *slight impairment of resonance*, as compared with the other side; the *respiratory murmur* is *bronchovesicular*, sometimes jerky in rhythm, and the *vocal resonance* and *fremitus* slightly increased or unaltered. Such physical signs are met with more frequently at the right apex than at the left, and oftener in the suprascapular fossa than anteriorly. The next most frequent seat is probably between the clavicle and second rib anteriorly. When phthisis sets in as a catarrhal pneumonia, consolidation is, as a rule, found at the apex. After convalescing from the attack, the patient has an area of consolidation usually at an apex of the lung: Over this area, in addition to the usual signs of consolidation (bronchial or feeble breathing, dulness, etc.), moist or dry subcrepitant *râles* are heard. As the case progresses there are fewer râles, but the signs of consolidation are still present, though there is no further exten-

sion of the process. Often after a cavity has been formed the disease is arrested or progresses very slowly. When softening begins, the *percussion-note* continues *dull* and the *breathing bronchial*; but it is often difficult to make out the quality of the breath-sounds because they are feeble and obscured by numerous *moist crackling râles* and *moist subcrepitant râles* from disintegration of lung tissue and bronchitis. After the patient has coughed several times and expectorated, and then takes a long breath, the quality of the breathing becomes perceptible. As the lung tissue is further softened and removed by expectoration cavities are formed. These, if large enough and superficial, give a *tympanitic note* on percussion, and, if there is communication with a bronchus, a *cracked-pot sound*. The *breath-sounds* are *hollow* and the *râles* are *bubbling and gurgling*, or large and mucous.

The normal vocal resonance is replaced by *bronchophony* and *pecto-riloquy*. Tactile *fremitus* may or may not be increased. (See Cavities.) But if the walls of the cavity are thick from indurated tissue, the percussion-note will be dull and the breathing bronchial. If the tissue composing the wall is less thick and dense, percussion produces a wooden sort of resonance. If much normal lung tissue intervenes, the percussion-note will be clear.

As tuberculosis of the lungs progresses, the clavicles and ribs become more and more prominent from the loss of fat, and *local flattening of the chest*, with *impaired expansion*, marks the seat of the disease.

Résumé of the Diagnostic Features of Pulmonary Tuberculosis. The striking phenomena of tuberculosis which are considered in the diagnosis are emaciation, anæmia, fever, cough, dyspnœa, chest pain, hemorrhage, the expectoration, and the objective symptoms. Of less diagnostic value, but important as collateral data, are the aspect, the occurrence of vomiting and diarrhœa, and of symptoms of secondary tuberculosis in other organs. Age and occupation may, to a certain extent, aid in the diagnosis.

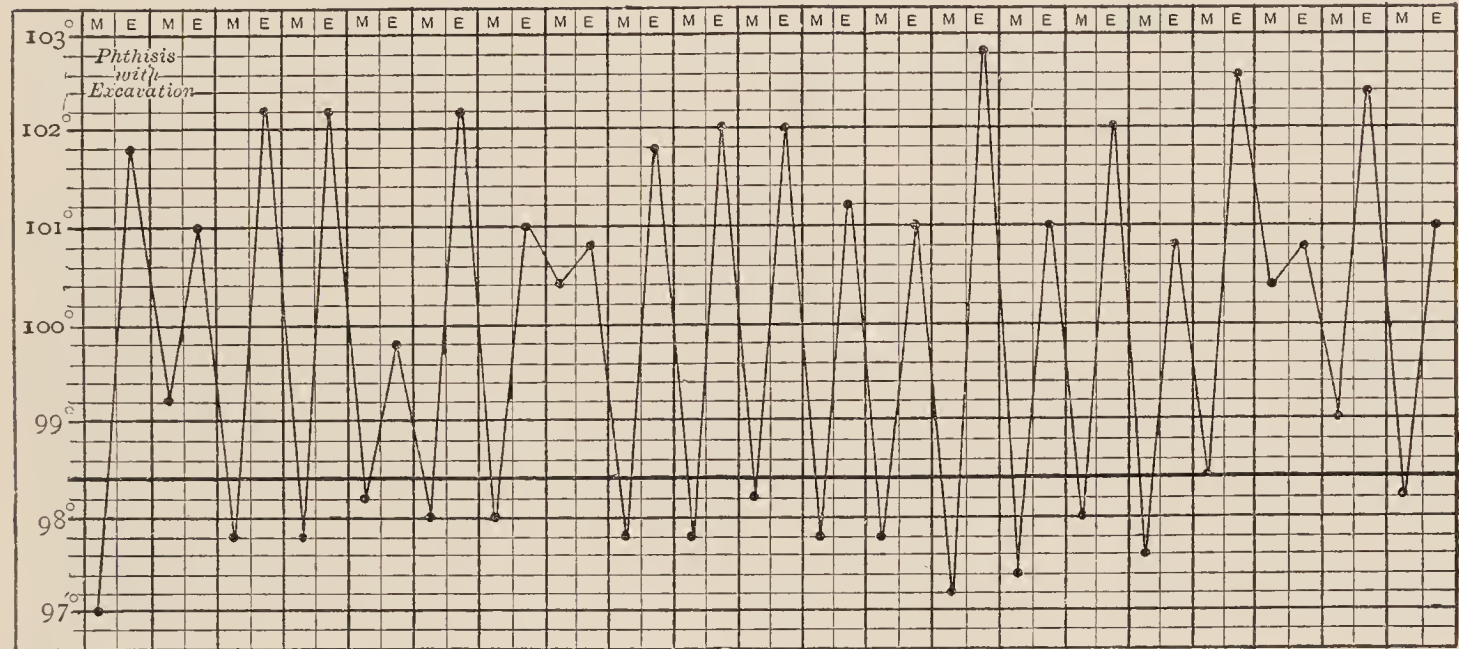
Symptoms. **EMACIATION.** This is always seen, even in acute forms of tuberculosis. It is rapid in the acute; slow and progressive in the chronic forms. In the latter there may be a temporary improvement in this respect. It must not be confounded with *muscular atrophy*, and the emaciation of *carcinoma*, *diabetes*, *anorexia nervosa*, and other exhausting diseases.

ANÆMIA. This is always pronounced. It may be associated with leucocytosis if there is cavity formation. The reduction of red cells and diminution of hæmoglobin are marked.

FEVER. This symptom is always present. The temperature should be taken every two hours for a time, to determine accurately the degree and course. It may be intermitting, remitting, or continuous. It may be intermitting in some acute forms, the morning fall reaching or going below normal. The difference between morning and evening temperature may not be more than a degree. In the acute form it is high and continuous, and soon may be attended by the typhoid state. In the more chronic cases it may be intermittent at first, then continuous, and finally intermittent again. In the later stages the intermitting fever is due to a

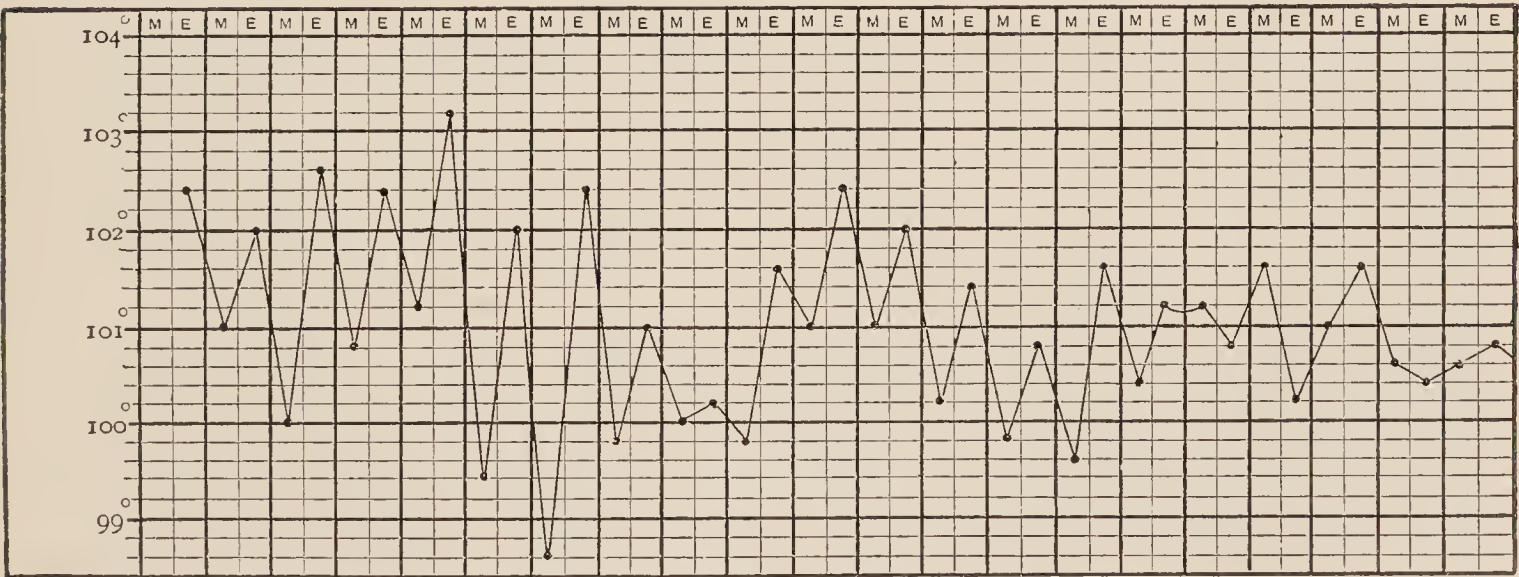
mixed infection, or sapræmia, due to the purulent contents (staphylococcus and streptococcus infection) of the lung cavities.¹ (See Fig. 345 and Fig. 346.) The intermittent fever of the early stages has frequently been mistaken for *malaria*. (See Fever.) The occurrence of fever in a patient who has been losing flesh, and is otherwise in poor health, excludes cancer and diabetes and other afebrile causes, and points strongly to tuberculosis. It must not be forgotten that in chronic tuberculosis in the aged the temperature may not rise above 100° F.; often, indeed, it is subnormal.

FIG. 345.



Intermitting fever of tuberculosis. (Original.)

FIG. 346.



Continued fever of tuberculosis. (Original.)

We must consider, therefore, that fever, the cause of which is not obvious, may be due to tuberculosis; and that if, when such probable causal conditions as gastro-intestinal catarrh or infectious disorders (malaria and syphilis) and suppurations are eliminated, the fever still persists, then the fever is probably of tuberculous origin.

¹ Leyden has pointed out that intermitting fever is part of the tuberculous process, and not a streptococcus or staphylococcus infection as formerly held, because pus micro-organisms are not found in the purulent contents of cavities, and because in other forms of tuberculosis, as empyema or joint-disease, they are notably absent, and yet such form of fever exists.—Deutsche med. Wochenschrift, Sept. 14, 1894.

SWEATS. Frequent sweating may be the first symptom complained of by the patient. It may occur with the tripod of symptoms of the intermitting febrile range—chill, fever, and sweat. It would be likely to occur at night under these circumstances. It may occur at any time, however. “Night-sweats” are alarming to the mind of the laity, and are really of diagnostic significance. The perspiration awakens the patient at night because it is so profuse. It may be only moderate, not rousing the patient until morning. It may be general or local. Local sweats are confined to the head and neck.

COUGH. Cough is one of the earliest symptoms. It may be the only symptom for some time. It is often dry and hacking at first, and may continue so for a long time. Later it is accompanied by mucoid and then mucopurulent sputa, which contain the characteristic elements. (See Sputum.)

DYSPNŒA. This is almost always present. The degree varies with the association of fever. When the latter is present, dyspnœa is more pronounced. It is more pronounced in acute cases. In miliary tuberculosis the frequency of respirations that attends the dyspnœa is out of all proportion to the physical signs. In this form cyanosis is more marked. In chronic localized phthisis the dyspnœa may occur only on exertion, after eating, or upon excitement. The bloodless lips may have a constant bluish hue. The fingers are dusky and become “clubbed.” In the later stages the dyspnœa is constant and in proportion to the extent of involvement of the lungs and the degree of fever. Although of diagnostic significance only when associated with other symptoms, it is most distressing, and is the cause of constant demand for relief.

CHEST PAIN. This is due to localized pleurisy or to myalgia. The latter may be seated in muscles strained by coughing. Pleuritic pains may occur in any situation, and vary in position from time to time. They may be due to extensive inflammation or to tuberculous pleurisy. Constantly recurring and unilateral chest pains, with or without signs of pleurisy, with cough and emaciation, are significant of the disorder under consideration. (See Pain.)

HEMORRHAGE. This symptom is alarming, and, in the large majority of cases, is due to pulmonary tuberculosis. It may mark the onset of the acute disease, and continue irregularly throughout its course or it may recur several times before the advent of the more common symptoms of the chronic form. It may occur at intervals of a few months or a year before emaciation, cough, and characteristic expectoration set in, or before bacilli are found in the sputum. Each attack is usually attended by fever, and is followed by anæmia and prostration. If hemorrhage of the lungs (see Symptoms) occurs in a young adult without cause (as aneurism or cardiac disease, etc.), it must be looked upon with suspicion. The likelihood of tuberculosis is increased if the bleeding occurs in a patient of tuberculous aspect in whom a family history of tuberculosis is found, or who has been exposed to infection. In the aged it may occur from a localized area of disease. Hemorrhage is also common in the late stages of tuberculosis. It is not at this period of diagnostic value as to the primary cause. It is usually due to erosion of an artery in a cavity.

Hemorrhage also occurs in tuberculosis during the quiescent period. The progress of the disease is arrested. The discharge of blood is accompanied by the expectoration of pulmonoliths, calculi formed by the degeneration of caseous areas.

VOMITING (see Gastro-intestinal Disease). This is a symptom which is often present in the early stages of tuberculosis of the lungs, and frequently masks the true condition. The vomiting may lead to the belief that a **LOCAL GASTRIC CATARRH** or **DIARRHŒA** is to blame for the general symptoms. The occurrence of fever with the gastric symptoms should lead to an examination of the lungs.

The occurrence of diarrhœa and symptoms of tuberculosis in other organs may thoroughly establish the diagnosis in tuberculosis of the lungs with otherwise obscure pulmonary symptoms. The intestinal discharges may contain tubercle bacilli, or they may be found in the urine, in joint-suppurations, or glandular enlargement.

THE SPUTUM (*q. v.*). The diagnosis is absolute when tubercle bacilli are found in the expectoration. Nummular sputa are more common in phthisical excavation. The sputum is discharged in tough coin-shaped masses, which sink when expectorated into a vessel containing water. Fragments of lung tissue (yellow elastic) point to tuberculosis, but are possible under other circumstances.

Physical Signs. The aspect of the patient is always suggestive, and is an aid to the recognition of the condition. The tuberculous or *phthisical chest*, the long neck and arms, the pale face, the occasional *hectic flush*, the *clubbed fingers*, the *emaciation* of the many subjects we see in our infirmaries, fix in our minds a composite picture the recognition of which goes far to diagnosticate the insidious disease.

The objective signs point to an invasion of air-containing structure by solid material, with collapse of lobules, leading to consolidation, followed by cavity formation, and in both stages by the occurrence of pleurisy. *Local contraction (flattening)* and *impaired movement* at an apex, with *inspiratory depression* above the clavicles, with suppressed breath-sounds and *prolonged expiration*, with *impaired resonance*, are the earliest signs of tuberculosis. In the chronic cases, contraction, impaired movement, dulness, and increased resistance from thickened pleura may override the signs of consolidation. No one physical sign is of diagnostic significance. The combination of signs, and the orderly procession by which they advance as the physical conditions progress, are the most diagnostic.

In the diagnosis of pulmonary tuberculosis the physical examination must be directed to a determination of the size of the lung, and of the extent of its expansion, by which we judge of the amount of air entering the lung, as well as to the presence of consolidation.

The tuberculous process is usually associated with diminution in the bulk of the lung. We can estimate the *size and the degree of expansion* by inspection, palpation, and percussion. The so-called diaphragm-phenomenon is studied and the *x-rays* employed. Any diminution in the excursion of the shadow of the diaphragm is evidence of diminished bulk of the lung or of diminished expansion. By palpation, with mensuration, measurements are taken. By percussion we estimate the lung boundaries.

The degree of expansion can be determined by securing the limits of liver-dulness and cardiac and splenic dulness in ordinary breathing, and then at the end of full inspiration and expiration. Valuable information is thus secured. Of course, in employing inspection and palpation the two sides of the lung must be compared. Percussion enables one to determine fairly early the presence of consolidation. In thin subjects the change in the note is more readily elicited than in fat or muscular subjects.

On auscultation in the early stage of tuberculosis roughness of respiratory murmur with prolonged expiration, feeble respiratory murmur, and jerking or cog-wheel respiration are common signs. These signs change gradually into bronchovesicular and then bronchial types of breathing. Crackling râles or clicking sounds and consonating râles attending these modifications of breath-sounds are of the greatest diagnostic importance. They must be brought out frequently by cough and then full inspiration.

THE SITE OF THE LESION. The situation of the physical signs is diagnostic. Percussion should be directed especially over those parts of the lung in which an infection is liable to occur, as the clavicular and subclavicular spaces, the anterior border of the upper lobe, the tongue-like part of the left upper lobe, which overlaps the heart, the supraspinous space, the upper interscapular region, and the upper borders of the lower lobes posteriorly. The latter is best secured by having the patient place the hand of the arm of the side percussed on the shoulder of the opposite side. The scapula is thus removed from the surface of the lung to be examined.

It is necessary also to consider carefully the general conditions. We inquire the age; adolescence and early adult life being the common periods in which pulmonary tuberculosis develops. The occupation, the history of exposure to the disease, the history of predisposition to tuberculosis in the family, the history of previous, now arrested, tuberculosis, as in joint-disease, or glandular tuberculosis (scrofula), are data deserving special consideration, as they may furnish corroborative evidence of the presence of the disease.

Diagnosis. The presence of tuberculosis is presumed in a patient with pulmonary symptoms—as a hereditary predisposition, abnormalities in the form of the chest, and imperfect development or hypoplasia of the circulatory organs. If the patient is under weight and has a poor appetite, and at the same time is undergoing unusual strain or anxiety, the possibility of tuberculosis is increased. Often before the physical signs of tuberculosis can be established, the shrewd physician will fear recurrence of tuberculosis if there are signs of anæmia, progressive loss of weight, slight fever, disturbed digestion, a frequent pulse, and persistent and localized bronchial catarrh. The examination of the lungs, the examination of the sputa, and the tuberculin test must be employed as soon and as often as practicable. (See Diagnosis of Tuberculosis,¹ Chapter I., Part II.)

The diagnosis is established by finding tubercle bacilli in the sputum.

¹ Several undoubted instances are recorded in which hospital residents and young physicians working in laboratories in which tuberculosis is studied, or constantly examining sputum, have been infected in the course of their studies.

Their absence, in spite of the most careful search, is against the tuberculous origin of the disease. (See Diagnosis of Tuberculosis, Chapter I., Part II.)

In other chapters the differential diagnosis of tuberculosis and other diseases will be pointed out. It must not be forgotten that the disease may set in as the terminal affection in many diseases. Thus, in diabetes, in insanity, in chronic cerebral or spinal disease, and in other affections, tuberculosis may develop insidiously, and finally cause death.

It must be distinguished from *chronic gastric disorders*, and particularly *anorexia nervosa*. It must not be confounded with *malaria*. It must be distinguished from *simple anæmia*, the cause of which may be recognized with difficulty. It must be distinguished from *chronic bronchitis* with bronchiectasis, from *pulmonary gangrene* and *carcinoma*. Finally, it must not be mistaken for *cancer of the œsophagus* and *aneurism of the aorta*, two divergent conditions which may have pulmonary symptoms simulating phthisis.

Gangrene of the Lung.

Gangrene is a rare disease of the lung, and, like abscess, always secondary. It may be produced by any cause which so obstructs the circulation that a portion of the lung dies in bulk. The gangrene may be circumscribed or diffuse: it results most frequently from pneumonia, but may be due to injury, to a general septic condition, or to embolism. It is rather frequently met with in the insane, possibly owing to particles of food which have found their way into the lung. Aspiration broncho-pneumonia, bronchiectatic and tuberculous cavities, sometimes lead to gangrene. Gangrene in the lung, as elsewhere, occurs in diabetes.

Symptoms. When it occurs in the insane or is of embolic origin, gangrene of the lung may remain latent, and in septicæmia it may be overlooked, on account of the general symptoms. In well-marked cases, however, the symptoms are characteristic. Symptoms and physical signs of pulmonary disease precede the specific symptoms of gangrene. With the onset of a moderate *fever*, *hæmoptysis* may occur at once or be preceded by the *expectoration* of a brownish purulent *sputa* having a most intense and persistent gangrenous odor. It contains fragments of lung tissue, altered blood, and putrid débris. (See Sputum.) It separates into the three characteristic layers in a conical glass. The *fetor of the breath* and the characteristic sputum are diagnostic.

Physical Signs.—The disease usually occupies the lower or middle lobe of the lung. The physical signs are those of *cavity*. The disease could with difficulty be distinguished from abscess were it not for the characteristic sputum, though in gangrene there is greater tendency to a general septic condition, with profuse sweats and collapse.

Abscess of the Lung.

Abscess of the lung may originate in causes outside the lung or in causes within the lung. To the former class belong those produced by suppurating bronchial glands, abscess of the mediastinum opening into

the lung, cancer of the œsophagus with ulceration, and abscess of the liver, suppurating hydatid cyst, or subdiaphragmatic abscess in general, bursting into the lung. Intrapulmonary causes are tubercle, septic emboli, in which case the abscesses are multiple and subpleural, and pneumonia. In the aspiration form of lobular pneumonia abscesses occur. Rarer causes are the presence of tumors and obstruction of the bronchi.

Abscess of the lung is therefore always secondary. Its *diagnosis* depends upon the demonstration of a *consolidation* in which a *cavity* subsequently forms, taken in connection with the history pointing to a definite cause. The *sputa* are copious, purulent, often odorless, sometimes offensive, but always without the fœtor of gangrene. They contain elastic fibre, but no bacilli except in tuberculous cases. (See Sputum.) In embolic abscess the signs of pleural friction can only be detected at times. Of course, the constitutional symptoms of suppuration are present.

THE DEGENERATIONS.

Emphysema of the Lung.

Emphysema consists in an “excessive, permanent, and unnatural distention of the air-cells,” or in “extravasation of air into the interlobular or subpleural cellular tissue.” (Laennec.)

Emphysema may be unilateral or bilateral. Local and unilateral forms are usually compensatory. Bilateral emphysema may be hypertrophic or atrophic.

It is more common in men than in women. Its symptoms are more common in childhood and after middle age. Two factors are essential in its causation. First, defective development of the elastic tissue of the lungs. Second, increased intra-alveolar air-pressure. The latter is due to a number of causes. In childhood, no doubt, nasal and nasopharyngeal obstructions are operative. In adults occupations which necessitate continuous and severe muscular effort, especially if coupled with forced expiration with closed glottis, act as causes. Such occupations are blacksmithing and playing upon wind instruments. Diseases which cause much coughing or respiratory effort, such as chronic bronchitis and whooping-cough, act in the same manner. Chronic mitral valvular disease and the lessened elasticity of the lung tissue of advancing age both favor congestion of the lung, and thereby predispose to emphysema. The disease is hereditary; several members of a family may be affected. It occurs in many in childhood, is in abeyance in adult life, and reappears in old age.

Symptoms. The prominent symptoms in hypertrophic emphysema are dyspnœa, cyanosis, and cough, with expectoration from associated bronchitis. There is no fever. The *dyspnœa* is in proportion to the degree of emphysema, and is aggravated by the coexistence of bronchitis, asthma, and eccentric hypertrophy of the right ventricle, which are very frequent complications in cases of long standing. When the degree of emphysema is only moderate, dyspnœa is not complained of except upon climbing or walking briskly, or after a hearty meal. But when the degree of em-

FIG. 1.—Anterior Aspect.



Hyperresonance. Enlargement of lungs and diminished respiratory movement of margins. Diminished fremitus. Signs of bronchitis.

physema is great, dyspnœa is constant; it interferes with all exertion, frequently necessitates orthopnœa, and prevents continuous speech, so that patients speak in broken sentences or syllables.

Cyanosis is marked. The livid lip is common in asylums for old men. The face is of a dingy pale color, but becomes bluish on exertion. The extremities are also dusky, and the blueness is general in severe cases. This cyanosis, the round shoulders, and the drawn, chronically anxious expression, if I may so term it, make it easy to pick out the emphysematous subjects in a ward of chronic cases.

Respiration is not accelerated, and may be diminished in frequency. It is often accompanied by *wheezing* when chronic bronchitis coexists.

The *cough* varies greatly in frequency; it may be altogether absent, since its presence simply indicates an associated bronchitis. This bronchitis may for years be present only in the winter. In children it may be associated with asthma. It may arise on changes of the weather; finally it becomes chronic. The expectoration is that of chronic bronchitis (*q. v.*). It is rarely stained with blood.

The general health suffers by loss of strength and capacity for physical and mental work, rather than by loss of flesh.

Physical Signs. (Plate XXIV.) The physical signs of emphysema depend upon its degree and upon whether it is complicated with chronic bronchitis or not.

Inspection. The patients are large-chested, stoop-shouldered, and short-breathed, and have an anxious expression of countenance. In well-marked cases the *chest* is *barrel-shaped*. (See under Inspection.) There is *little movement of the chest* in respiration, because the lung is already in a condition of full inspiration (expiratory dyspnœa).

Palpation. *Vocal fremitus* is usually diminished.

Percussion. The *percussion-note* is abnormally *clear* and may even be *tympanitic*. *Hyper-resonance* is typical of the disease. When the distention is extreme, the note may be woodeny. The lungs are enlarged. The *heart-dulness* becomes *obliterated* by the overlapping lung. The upper margin of the liver falls one or two interspaces below normal. The resonance extends higher above the clavicles than normal.

Auscultation. The *inspiration* is found to be *distant* and *feebler* than normal, while the *expiration* is *prolonged*, and may become three or four times the length of the inspiration. *Grazing* or *rubbing sounds* have been described and attributed to the friction of distended vesicles against the pleura. Other adventitious sounds are due to an associated bronchitis, pleurisy, or tuberculosis. But bronchitis is such a common accompaniment of emphysema that the *râles* of the former become almost symptomatic of the latter. Their character in emphysema does not differ from that in chronic bronchitis (*q. v.*).

Physical Signs of the Heart. The *apex-beat* is *absent*. There is *epigastric pulsation* or systolic shock. The normal area of heart-dulness is encroached upon by the distended lung, and the *heart* itself is *pushed to the right*, the apex-beat being frequently at the xiphoid cartilage. If the emphysema attains a very high degree, there may be no perceptible dulness, except on very strong percussion over the cardiac region. The

heart-sounds appear *feebler* and *more distant* than normal. The right ventricle becomes dilated and hypertrophied as the result of the pulmonary congestion produced by emphysema. The *pulmonary second sound* is *accentuated*. A *tricuspid regurgitant murmur* may be heard. Venous congestions are common in the later stages. Albuminuria is common. Edema of the feet and limbs may occur, but general anasarca is rare.

Diagnosis. This is based upon the history (heredity, occupation, long duration), the occurrence of dyspnoea and cyanosis, and of winter cough or chronic bronchitis, and upon the physical signs.

Emphysema can be distinguished from *pleural effusion* and from *aneurism*, which may cause dyspnoea, by the universal hyper-resonance on percussion. Pleural effusion, which also causes bulging, is usually unilateral, and the percussion-note is flat. The area of dulness of the heart and aorta is diminished in emphysema.

Pneumothorax, which most resembles emphysema in its physical signs, develops suddenly, affects one side, and gives a hollow, tympanitic note on percussion. The succussion-splash, metallic tinkling, and coin-test have no counterpart in emphysema; moreover, the antecedent history and mode of development are different.

Atrophic emphysema is due to the degeneration of age. The lung is reduced in size, the diameters of the chest are lessened, the ribs are oblique, and there is atrophy of the chest muscles. The patients have dyspnoea and exhibit other signs of senility.

In *interlobular emphysema* the physical signs are the same as those of vesicular emphysema, but it develops suddenly and is liable to be followed by emphysema (intercellular) of the neck, which on palpation gives a peculiar crepitation. The friction-sound and crackling which have been described as occasional adventitious sounds in vesicular emphysema are more commonly heard in the interlobular form. It is caused by rupture of the air-cells, and hence occurs in diseases in which a great strain is put upon them—especially in whooping-cough, but also occasionally in pulmonary hemorrhage and pneumonia; violent coughing and laughing, and great straining, as in child-labor, are capable of producing it.

Bronchiectasis.

Dilatation of the bronchi occurs secondarily to affections which tend to weaken the walls of the tubes and to lessen their elasticity. Hence it is found in chronic bronchitis with emphysema, in chronic phthisis, in catarrhal pneumonia in children, in chronic obstruction from external pressure or foreign bodies. (See Obstructions.) It also occurs when the lungs contract in fibroid pneumonia or in pleural thickening. It occurs in two principal forms: the *simple*, in which the affected tubes are uniformly dilated; and the *saccular*, in which larger or smaller pouches are formed. It is commoner in males than in females, and probably begins most frequently in adult or middle life. One lung only is affected in about one-half the cases, and when both lungs are affected (chronic bronchitis and emphysema), it is not often to the same degree.

Subjective Symptoms. These consist of cough, expectoration, and a

FIG. 1.—Anterior Aspect.



Chronic pleurisy with induration of the right lower lobe and bronchiectasis.
Vicarious emphysema of the left lung. Bronchitis.

variable amount of dyspnœa. Eventually there may be some loss of flesh and strength.

The *cough* is usually paroxysmal. It may occur only in the morning after the dilated tube fills and it may follow change in position. A paroxysm is followed by copious expectoration, sometimes amounting to a pint and a half in twenty-four hours. The *sputa* are grayish brown and mucopurulent, faintly or extremely fetid. They contain mucus, pus, casts of the tubules, and various salts. Charcot-Leyden and fatty crystals, vibrios, leptothrix, and bacteria (Fox) can be found on microscopical examination. Elastic fibres are found only if the tubes are ulcerated. In a conical glass the sputum separates into three layers—a frothy brown top, a thin mucoid layer in the middle, and a granular layer below. Hemorrhage may occur periodically even when tuberculosis is absent. It was a feature in 14 of the 35 cases reported by Fowler and Godlee. *Dyspnœa* is not usually severe, except when the dilatation is complicated by disease of the heart or lungs, or by an acute attack of bronchitis.

Physical Signs. (Plate XXV.) The physical signs differ according to the extent and variety of the dilatation. In simple dilatation there may be nothing different from the signs found in chronic bronchitis, except a tendency to more *bronchial respiration*, with *râles* having a metallic quality. Percussion will vary according to the degree of alteration of the lung tissue surrounding the affected bronchi, and according to the extent of the dilatation and its proximity to the surface. In the *simple forms* the *percussion-note*, if altered, is somewhat *less resonant* and *higher in pitch*, whereas in *saccular dilatations*, favorably situated for percussion, the note is *tympanitic* if the pouch is empty. On auscultation in simple dilatation the *breathing* approaches the *bronchial* and is accompanied by bronchial *râles*. In saccular dilatation the sounds are practically those of a cavity, respiration varying from bronchial to *amphoric*. *Vocal resonance* and *tactile fremitus* are usually both *increased*, but the latter may be diminished.

Diagnosis. The diagnosis of simple dilatation from *chronic bronchitis* may be impossible, but copious and fetid expectoration indicates the former. The diagnosis of the saccular form from *tuberculosis* of the lung *with cavity* is difficult. Wilson Fox says that severe cases are usually associated with consolidation of the lung or with tubercle; but even without the presence of the latter they often present phthisical symptoms—retraction of the chest, with the physical signs of excavation, pains in the side, hæmoptysis, pyrexia, nocturnal perspiration, and diarrhœa—which may all coexist with only an induration of the lung and dilatation of the bronchi. The diagnosis must be made by noting the persistence of the physical signs, which change but little and are not progressive as are those of tuberculosis; the protracted course of the disease; the character of the sputum, and the comparatively slight impairment of the general health.

The Morbid Growths of the Lungs.

Cancer of the Lungs. The new growth may be primary or secondary. The latter is most common. Of primary cancer, the epithelioma is most

common; encephaloid and scirrhus come next. Secondary new growths succeed disease in the abdominal organs, the genito-urinary tract, the bones, the breast, and the eye.

Symptoms. The general symptoms of carcinoma accompany the thoracic symptoms. *Chest pain, dyspnœa, cough*, and a peculiar expectoration belong to the latter. The pain is due to associated pleurisy; the dyspnœa is paroxysmal. (See Dyspnœa from Pressure on Bronchi.) The *expectoration* is dark like prune-juice. Signs of intrathoracic pressure are seen. The external thoracic veins are enlarged. The face and arms may be cyanosed, or one arm only may be affected. The heart may be dislocated, the trachea changed in its course; compression of trachea and bronchus causes dyspnœa.

Physical Signs. In primary cancer the affection is unilateral; in secondary forms, bilateral. The *physical signs* are those of *pleural effusion* or of local *consolidation*. The consolidation may be massive and not partake of the shape of a lobe. Often signs of effusion and consolidation are combined (enlargement, immobility, absent fremitus, but bronchial breathing). In the secondary forms the disease is bilateral. The signs are mixed. They indicate diminished air in the lung structure. Care must be taken not to overlook the pleural effusion which accompanies the process, the removal of which gives temporary relief. In both forms external lymphatic glands, particularly the cervical, may be enlarged.

Diagnosis. The diagnosis is based upon: (1) the age (after forty); (2) the occurrence of emaciation; (3) the duration of the disease, often rapid, rarely beyond eight months; (4) the presence of primary disease elsewhere; (5) the presence of moderate fever; (6) the signs of intrathoracic pressure; (7) the involvement of lymphatic glands; (8) the occurrence of irregular areas of consolidation and of pleural effusion, alone or combined; (9) the characteristic expectoration; (10) dyspnœa due to pressure on the bronchus or trachea; (11) the absence of bacilli from the sputum.

An effusion can often be recognized only after puncture. Hæmothorax is not necessarily present.

Sarcoma. This is sometimes primary, but more often secondary. The *symptoms* and the *physical signs* are much the same as in carcinoma. The age (usually under forty) may be an aid in *diagnosis*.

Gross Parasites of the Lungs.

Hydatid Disease of the Lungs. The lungs are affected in about 11 per cent. of the cases of hydatid disease.

Symptoms. According to Wilson Fox, these consist of *dyspnœa, pain in the chest, cough*, occasional *hæmoptysis*, and sometimes the *expectoration* of hydatids, the *sputa* being otherwise bronchitic, or presenting the characteristics of pneumonia or gangrene when these complications are present. Gradually *weakness* increases, sometimes with *pyrexia*, which, when combined with *emaciation*, may impart to the case a considerable resemblance to phthisis. *Pressure-symptoms* occasionally occur.

Physical Signs. These are either those of *consolidation* of the lung or of *pleural effusion*, together with certain peculiarities depending on the

size and site of the tumor. Graham states that they are more frequent in the right lung and more common at the base, causing marked bulging of the thoracic wall. When the physical signs are those of pleural effusion, localization of the fluid to a definite area takes place, and hence is not related to the shape of the pleural cavity. The *breathing* may be *tubular*; there is condensed lung between the hydatid and the thoracic wall.

Diagnosis. The symptoms present—cough, dyspnœa, anæmia, emaciation, and clubbing of fingers—too often lead to the diagnosis of *phthisis*. Hæmoptysis occurs in many cases. The temperature is normal—an important point in diagnosis. If the cyst ruptures, the sputum is diagnostic. Complications often mask the diagnosis. It must be distinguished from *pleurisy*, localized *empyema*, *pulmonary abscess*, *phthisis*, *actinomycosis*, and *mediastinal tumors*.

DISEASES OF THE PLEURA.

The large lymph-structures which cover the lung and line the inside of the thorax are often the seat of disease. It is usually of an inflammatory nature.

Acute Pleurisy.

Pleurisy, or pleuritis, is the most common affection of the pleura. It may be, as to its distribution, bilateral or unilateral; as to its extent, local or general; as to the nature of the inflammation, plastic, serous, or purulent. The inflammation is rarely primary. It may arise in the course of general disease, or it may be the result of the extension of inflammation, chiefly of an infectious nature, from neighboring structures.

1. *Disease of the ribs or vertebræ, of the mediastinum, of the aorta, and of the œsophagus*, gives rise to various forms of pleurisy, depending upon the nature of the primary affection.

2. *Diseases below the diaphragm; abscess of the liver; perforative inflammation of other viscera adjacent to the diaphragm; abscess of the spleen or pancreas; pus in the pelvis or about the appendix*, may give rise to purulent pleurisy by the pus burrowing upward or by infection through the lymph-channels.

3. *Disease of the lungs.* In the large majority of cases pleurisy in some form occurs in the course of pulmonary disease. In all surface inflammations of the lungs there is associated pleurisy. It is seen in pneumonia, in tuberculosis, in gangrene, and in abscess.

Pleurisy may be *simple* or *purulent*. Empyema is always due to infection from the exterior, as the ribs; from the lungs (pneumonia); supuration below the diaphragm; or to general infective processes, as septicæmia, pyæmia, and tuberculosis.

The *general diseases* in the course of which pleuritis arises are usually infective, or of such nature as to cause irritating products to circulate in the blood. Of the former, the most common is tuberculosis; the next most common are septicæmia and scarlatina; while to the latter class belong Bright's disease, gout, diabetes, rheumatism, and scurvy. Puru-

lent pleurisy is more common in children than in adults; in males than in females; and more common in tuberculous pleurisy and pyæmia than in rheumatism and Bright's disease.

Three stages usually occur in acute pleurisy, although the morbid process may be arrested in the first stage.

The First Stage—Dry Pleurisy.

Symptoms. The onset of the disease is usually abrupt, and is marked by *fever*, which may or may not be preceded by *chill*, and is followed by pain in the side, *dyspnœa*, and *cough*. The *pain* is sharp, stabbing, or tearing in character, and is usually, but not always, referred to the seat of pleurisy. This is most frequently on a level with the nipple, or a little below this, and more often anteriorly or in the axilla than posteriorly. The pain is caused by the rubbing together of the inflamed surfaces of the pleura, and hence is excited by respiration and cough. For this reason the patient is inclined to *restrict the motion* of the affected side as much as possible; he does this by leaning over toward that side and by pressing his elbow in against the chest-wall. Pain is usually the first symptom noticed by the patient. The *cough* is dry and painful. Fever is moderate.

Physical Signs. The physical sign in primary cases is a *friction-sound* heard on inspiration and expiration. This friction-sound may be a nest of fine, dry, crepitant râles, which are very superficial, and appear to be just under the ear; or a coarse rubbing sound, heard over a larger surface, and resembling a bronchial rhonchus, from which it can be distinguished by its persistence after the patient has coughed. The lungs themselves present nothing abnormal.

If the inflamed surfaces become glued together by plastic lymph, recovery usually occurs very soon, though pain often persists for a long time in lessened degree, and the pleurisy is liable to be relighted.

The Second Stage, or Stage of Effusion. When effusion takes place, the two layers of the pleura become separated; hence pain and friction-sound cease, and physical exploration shows that a collection of fluid intervenes between the chest-wall and the lung.

Symptoms. During this stage there occur slight or moderate *fever*, sometimes intermittent in character, with recurrent *chills*; considerable *dyspnœa*, occasionally amounting to *orthopnœa* when the effusion is very extensive; and dry *cough*, which adds greatly to the dyspnœa. There is frequently some evidence of insufficient oxygenation of the blood; when this amounts to *cyanosis*, the condition is one of great danger. The *urine* presents changes in amount; in advancing effusion lessening very much; but increasing in amount with the decline of the fluid. Pleurisy may be complicated with bronchitis, pneumonia, and pericarditis.

Physical Signs. (Plates XXVI. and XXVII.) In this stage are seen (1) *enlargement of the affected side*, increase in semicircumference, with fulness of interspaces; (2) *diminution of movement*; (3) *absence of vocal fremitus and resonance*; (4) *dulness or flatness* (deadness) on percussion, with *great increase in the resistance* to the pleximeter finger; (5) *absent or greatly diminished respiratory murmur*; (6) *displacement of organs*.

The dead *percussion-note* being caused by fluid, it follows that its upper

PLATE XXVI.

FIG. 1.—Anterior Aspect.

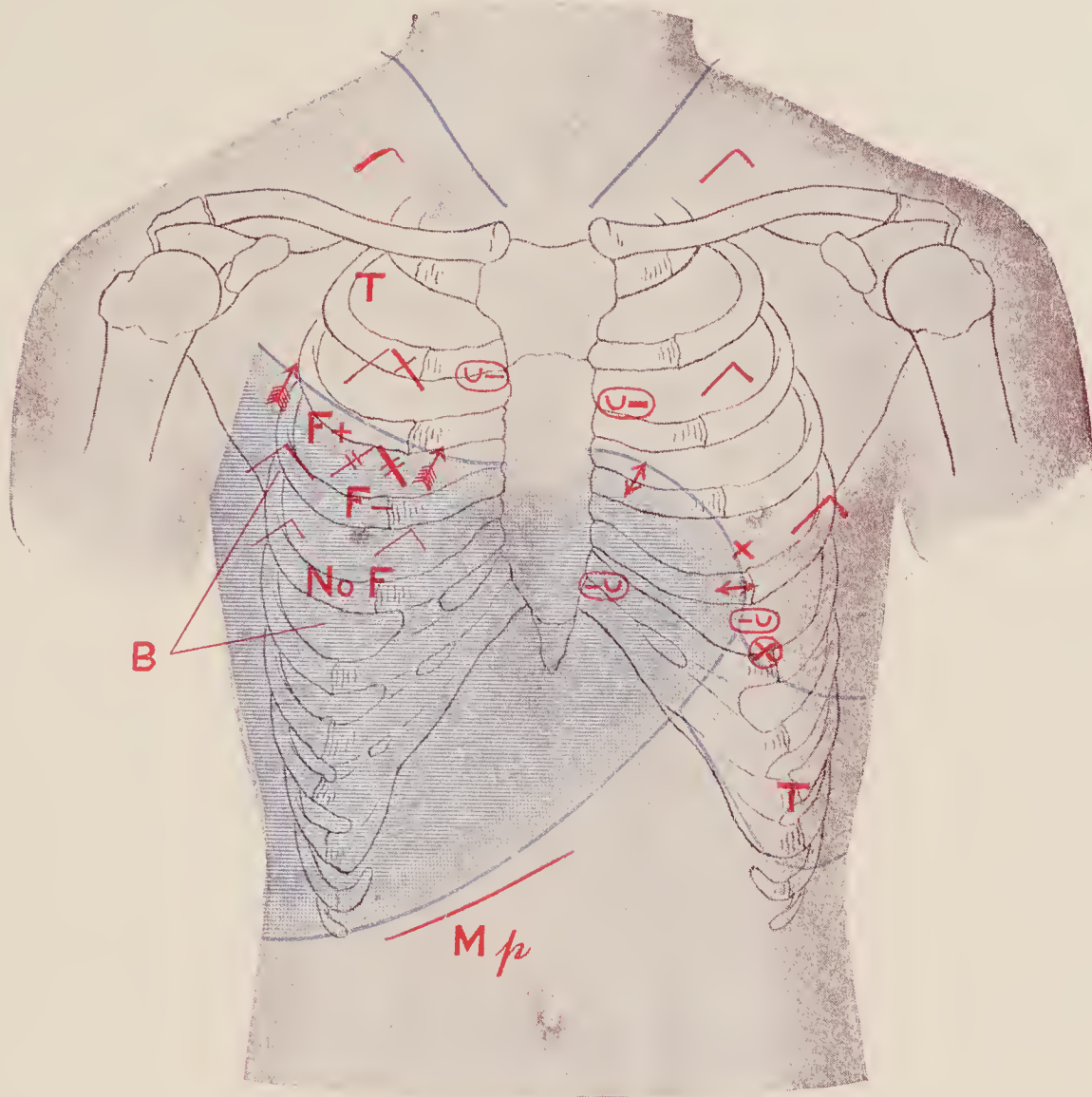
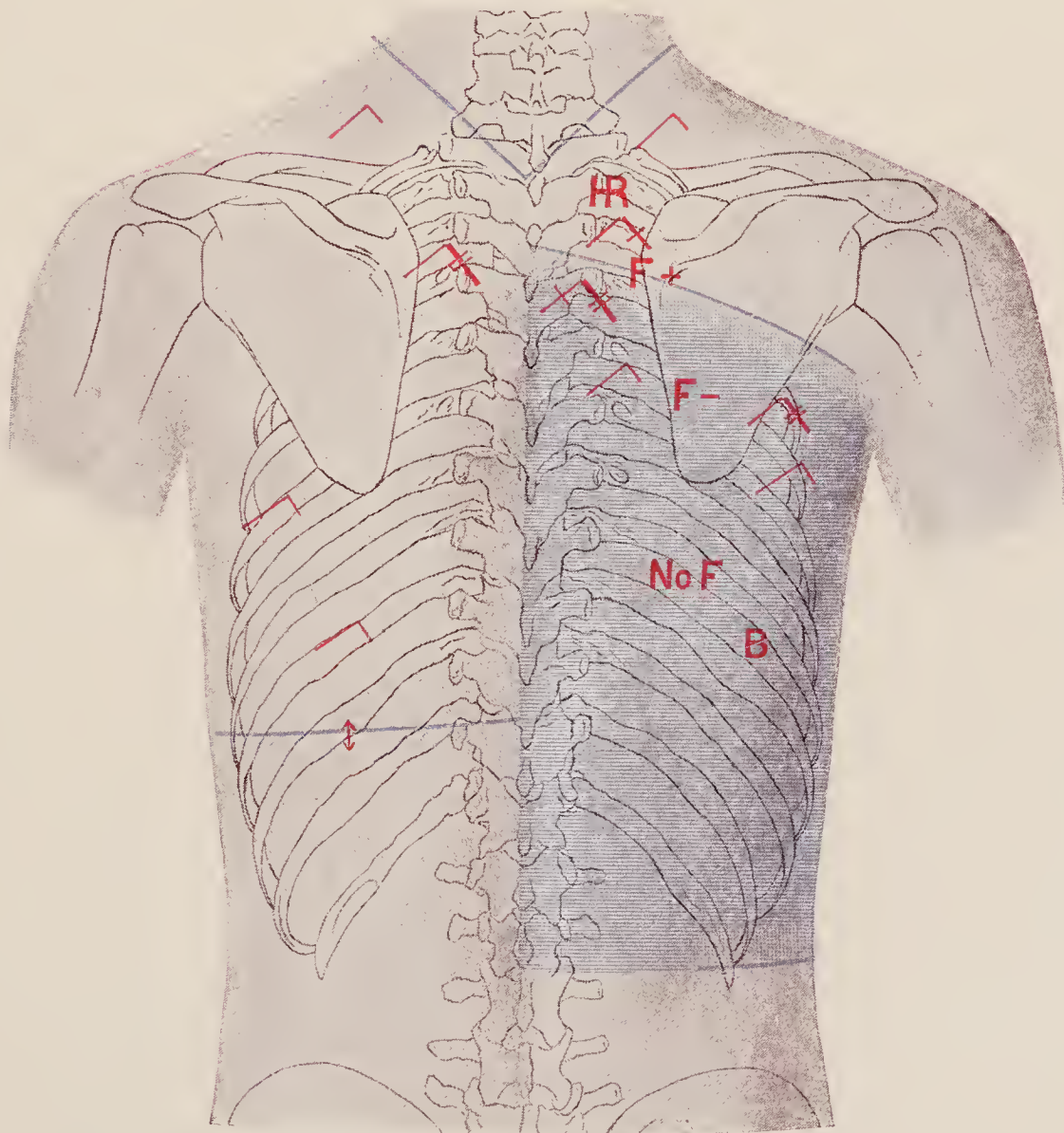


FIG. 2.—Posterior Aspect.



Pleurisy with Effusion (right-sided).

PLATE XXVII.

FIG. 1.—Anterior Aspect.

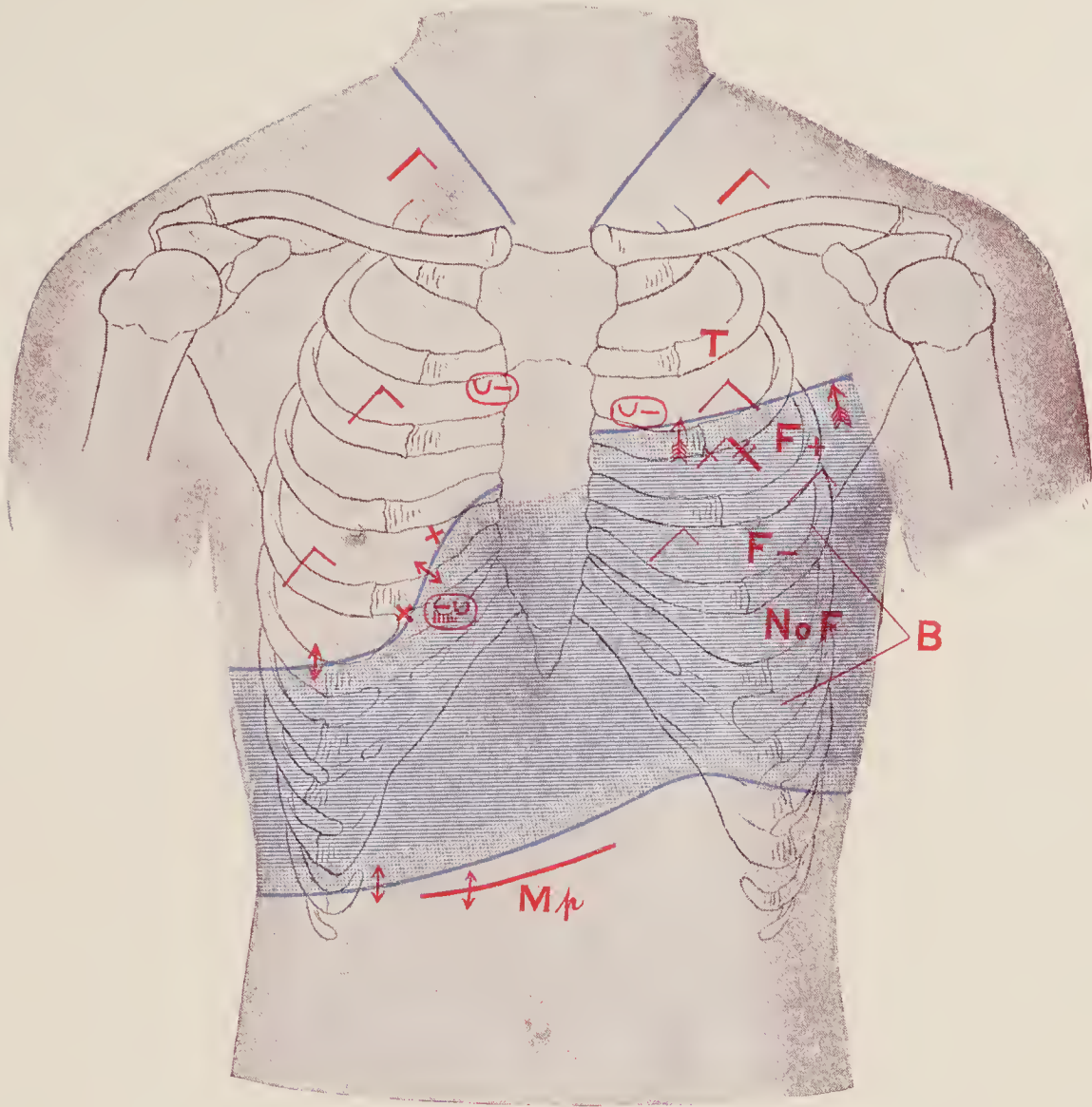
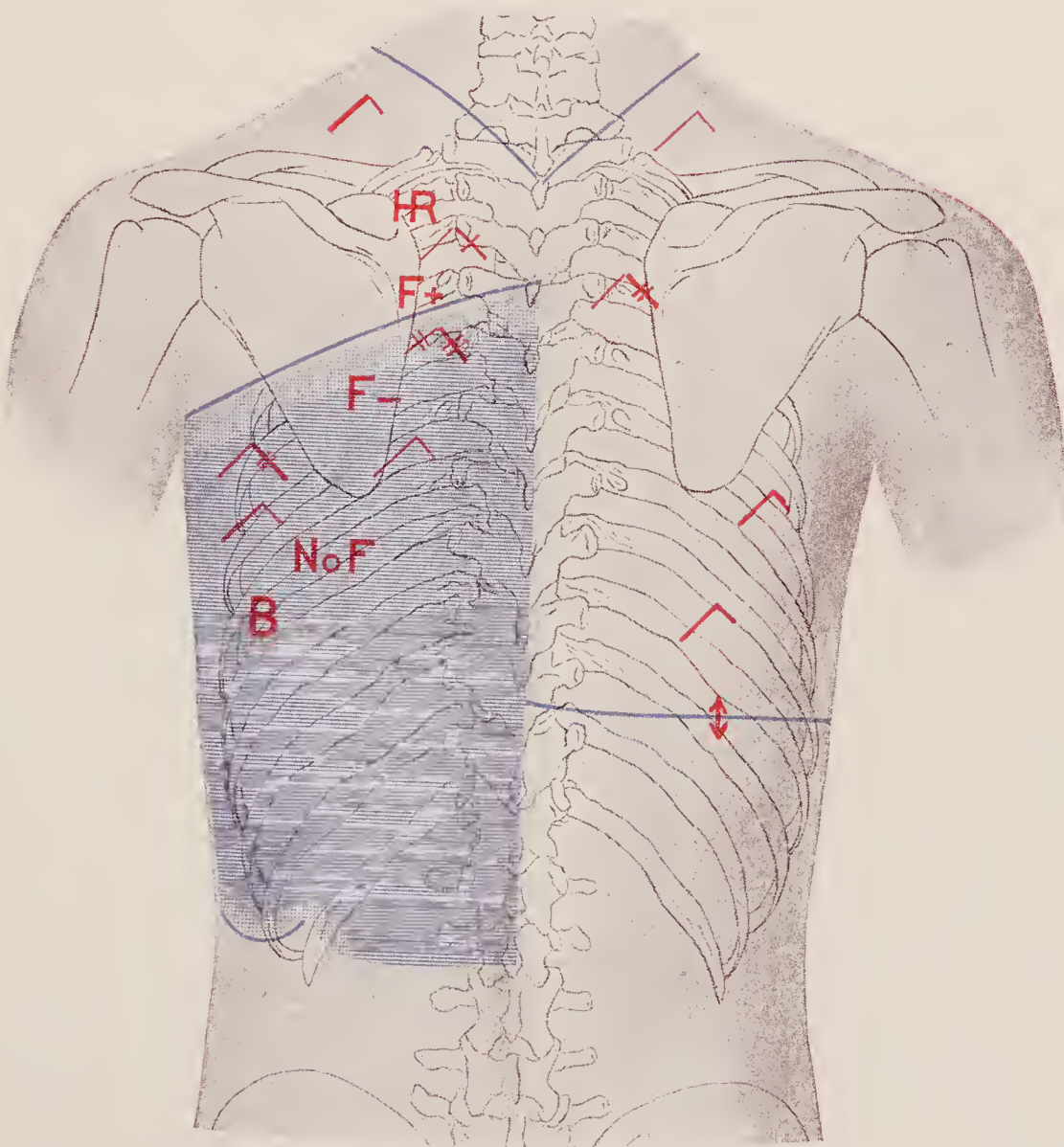


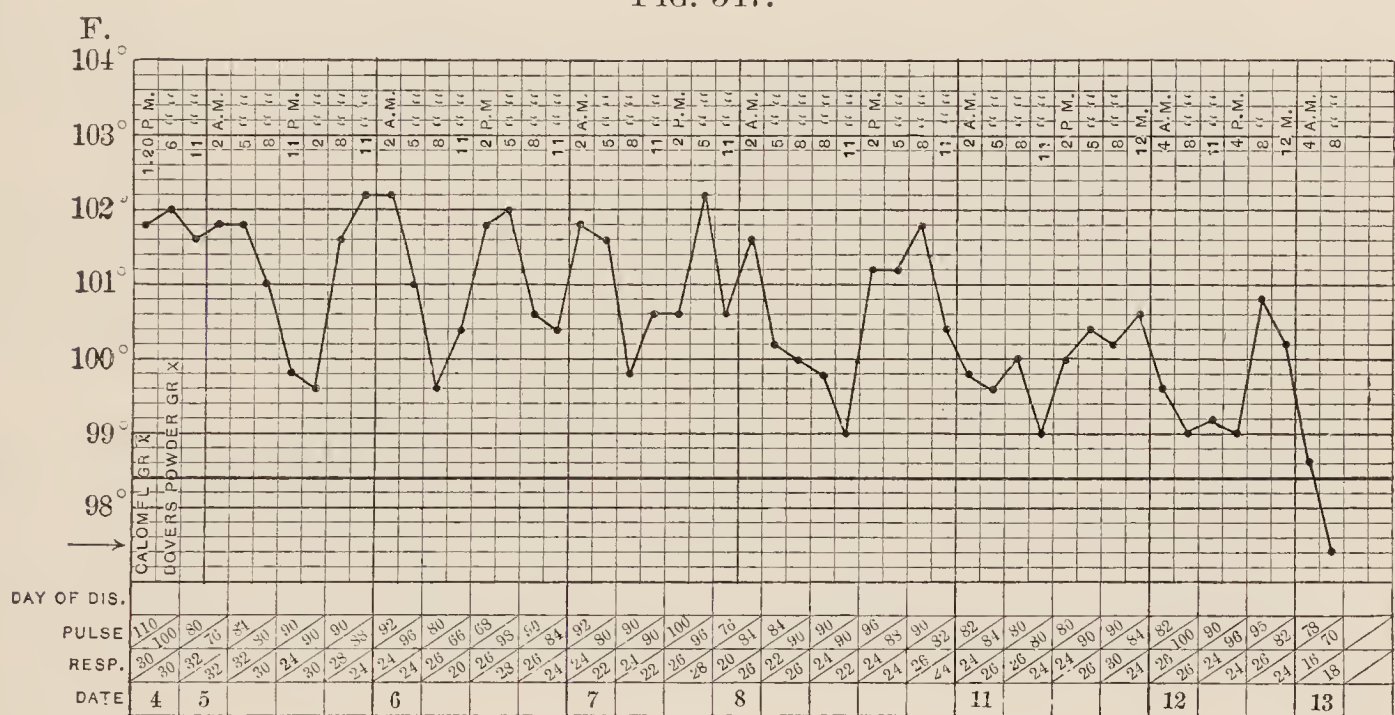
FIG. 2.—Posterior Aspect.



Pleurisy with Effusion (left-sided).

level will change with the position of the patient if the fluid is free. If the upper level is at the third interspace when the patient is sitting up, it will fall to the fourth or lower when he is lying down. This change of level can not be appreciated when the effusion is very large. Moreover, above the line of dulness the percussion-note is hyper-resonant or tympanitic—*Skoda's resonance*. Toward the spine on the affected side there may be partial resonance and bronchial breathing, because here the lung is compressed against the vertebræ. In large effusions the tympanitic resonance in the second interspace does not change when the mouth is opened—that is, “Williams' tracheal tone” can often be elicited. The upper limit of dulness in large pleural effusions is higher at the spine and slopes downward, and is lowest in front. This parabolic line is only obtained when the patient is in the erect posture. In moderate effusions

FIG. 347.



Pleurisy with effusion. Recovery. (Two days omitted.) (Original.)

the line of dulness is lowest near the spinal column, rises in the middle of the scapula and slopes downward, assuming the shape of the letter S as it passes toward the front. (Garland.) The patient should take deep breaths before the percussion is performed. At the left base in front the semilunar space is obliterated, dulness continuing to the margin of the ribs. In small effusions the dulness may be limited by the posterior axillary line, resonance being present in the lateral and anterior regions.

On *auscultation* below the upper level of the effusion, posteriorly, the voice frequently has a metallic quality resembling the bleating of a goat—*ægophony*. It occurs usually when the effusion is moderate, and may be heard only over a limited area. It is commonly heard at or above the angle of the scapula. Bronchophony may be heard when tubular breathing is present.

While the respiratory murmur is, as a rule, absent, breath-sounds may be heard, and are then weak and distant, or bronchial. In such cases there may be adhesions. Bronchial breathing may be present along the spine in small effusions, and in large effusions in the interscapular region. Bronchial breathing, tubular in character, is said to be almost constant in

children. It may also occur when pneumonia coexists. In one of the cases in my ward the signs were like those of a large cavity at the right base, but the immobility, the absent fremitus, the enlargement, and the exploratory puncture disproved its presence.

At the level of the fluid a friction-sound may persist. Above the level of fluid anteriorly, the breath-sound may be bronchial or bronchovesicular, associated sometimes with fine râles, due to compression and slight œdema.

DISPLACEMENT OF ORGANS. If the effusion is on the left side, the mediastinum and heart become displaced to the right, and the apex-beat may be found in the epigastrium or even to the right of it. The occurrence of displacement of the heart must also be judged by the position of maximum intensity of the heart-sounds, as the heart may be behind the sternum. At the same time the semilunar space (Traube's line) is lower than usual or entirely effaced. On the left side inaction of the diaphragm may be observed, and the tissues at the costal margin fall in with each inspiration. If the effusion is on the right side, the diaphragm, and with it the liver, is depressed, and the mediastinal contents are moved to the left.

Empyema. The above-mentioned physical signs apply chiefly to serous effusions. They are also present in effusions of pus. Other physical phenomena, however, and different general symptoms distinguish the two kinds of effusions, although it must be confessed that aspiration must often be resorted to before a positive diagnosis can be made.

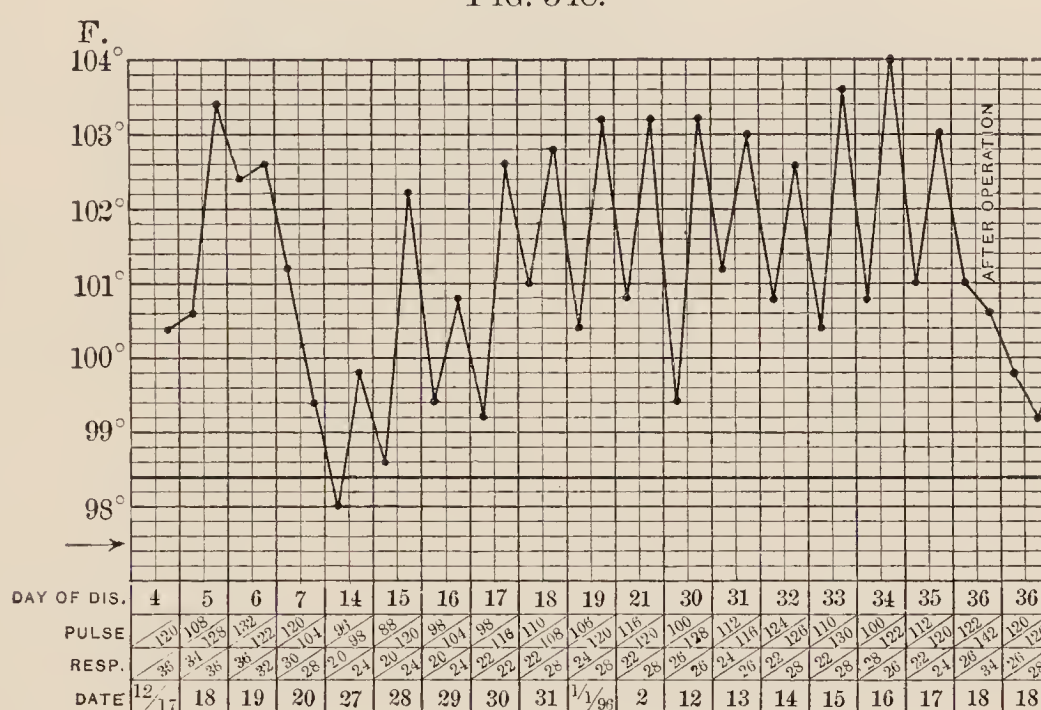
Symptoms. The general symptoms are more marked in empyema than in simple serous effusion. The *temperature* is higher from the onset. It soon becomes intermittent or remittent. *Chills* or chilliness may attend the beginning of each febrile paroxysm, and *sweats* occur with the daily fall of temperature, or at irregular periods during the twenty-four hours. The *heart's action* is more rapid and the *pulse* more feeble, soon becoming dicrotic. Examination of the *urine* may aid in the distinction of the two forms of the effusion. *Albumosuria* occurs in purulent pleurisy. It must be remembered that albumosuria occurs in suppuration from other causes. Thus, in phthisis with suppuration of a cavity, pleural effusion may develop. The albumosuria that attends the primary process must not be mistaken for that which occurs in empyema. *Indican* is also present in excess in the urine in suppuration. Before a positive conclusion is arrived at, two or more examinations of the urine should be made. Examination of the *blood* may aid in arriving at a conclusion. In purulent effusion there is usually leucocytosis.

Physical Signs. The physical signs of empyema are the same as those of other effusions within the pleura. In addition, especially in children, local *œdema* of the chest-wall may be found. Another sign was pointed out by Bacelli, and is held by others to be of diagnostic significance. In purulent effusions the fremitus produced by the *whispering voice* is not transmitted to the hand laid over the effusion, whereas in serous effusions such vibrations are transmitted. In lobulated empyema the diagnosis is very difficult. In one of my cases dulness continuous with that of the heart extended to the second rib and laterally to the post-axillary line. The dulness occupied three interspaces. Additional physical signs were

immobility, prominence of interspaces, localized above the heart, absent fremitus and resonance. There were no breath-sounds, but an abundance of râles, apparently very superficial. The râles complicated the physical signs. Martin removed 2 ounces of pus from a small abscess above the heart and between the lobes.

In empyema a local area may become more prominent and the surface assume an inflammatory appearance. It is an indication of discharge of the abscess through the chest-wall. It is usually found in the fifth interspace in front, or below the angle of the scapula behind—*empyema necessitatis*. (For a microscopical and chemical description of the "Effusion within the Pleural Sac," and of the morphological elements of the purulent effusions, see Chapter XLV., Part I.)

FIG. 348.



Empyema following pneumonia. (Fever absent from seventh to fourteenth day.) (Original.)

Notwithstanding the positive physical signs of effusion the character of the effusion may not be recognized until perforation into a bronchus has taken place. The peculiar character of the expectoration that attends this accident is described in the section on Sputum.

Hydrothorax. This is an accumulation resulting from a transudate. (For character of the fluid, see Chapter XLV., Part I.) It occurs in the course of diseases which produce anasarca, as failing organic heart disease, chronic Bright's disease, and debilitating diseases, as scurvy. Locally, it may attend carcinoma of the pleura or obstructive disease of vessels within the mediastinum.

Symptoms. The general symptoms belong to the primary disorder. *Dyspnœa* may develop gradually and even amount to orthopnœa. It is distinguished from inflammatory effusions by the character of the fluid, by the absence of the general symptoms of inflammation, by its insidious development, and by its bilateral distribution.

Physical Signs. The physical signs of hydrothorax are those of *effusion* in acute pleurisy.

Hæmothorax. The transudation of blood into the cavity of the pleura occurs rarely from the rupture of an aneurism into the sac. The fluid is then pure blood. Serous effusions in which a large amount of

blood is found point to primary carcinoma of the pleura, or to tuberculous disease. Both specific processes of this serous membrane may occur, however, without transudation of serobloody fluid.

Thickened Pleura. Chronic inflammation, with thickening of the pleura from excessive development of connective tissue, occurs in tuberculosis and in cases of combined pleuritis and peritonitis. The thickening of the pleura is usually more marked at the base.

Subjective symptoms of cough and dyspnœa are present. The degree of *cough* depends upon the condition of the lung. If there is bronchitis or tuberculosis, the cough is excessive. The amount of *dyspnœa* depends upon the degree of compression of the lung by the thickened pleura.

Physical Signs. (Plate XXVIII.) These are pronounced, and are those of effusion, but without enlargement of the chest. There are marked *contraction* and *diminution in movement* of the affected side. The *fremitus* is *absent*. There is *dulness* on percussion, or even *flatness*. The *breath-sounds* are *distant* or are *absent*. Along the vertebræ, especially opposite the angle of the scapula, *bronchial breathing* may be heard.

Tuberculous Pleurisy. The affection may be acute or chronic. It may occur primarily, it may be a part of general tuberculous infection, or it may occur secondarily to disease of the lungs. It may give rise to all forms of the inflammatory process, to (1) dry pleurisy; (2) pleurisy with effusion; (3) pleurisy with great thickening. Often the distinction between tuberculous pleurisy and pleurisy due to other causes can not be determined positively. If the pleurisy be associated with tuberculosis in other organs, or if the patient be of tuberculous habit and be exposed to infection, or if there has been a history of previous tuberculosis, the pleuritic infection is probably of tuberculous origin. If the affection is bilateral and associated with peritoneal inflammation, and if at the same time no other cause exists for serous membrane inflammation, the probability of its tuberculous origin is very strong.

Pulsating Pleural Effusion. Wilson has made the most recent studies of this rare affection. The effusion within the pleura pulsates synchronously with the ventricular systole; the pulsation is detected usually by inspection and palpation. In some instances its presence is only determined by palpation. It may be confined to two or three interspaces, or occupy the anterior aspect of the thorax and the axillary region on the left side. Rarely the pulsation is behind. It is usually situated on the left side. The original effusion is purulent in the large majority of cases. The physical signs and general symptoms of empyema are present. Nevertheless, the disease simulates aneurism of the aorta. The latter affection, however, is accompanied by vascular symptoms and physical signs in the course of the aorta. Pulsating empyema is distinct in movement from the pulsation of the aorta and occupies a different anatomical site.

Diaphragmatic Pleurisy.

In diaphragmatic pleurisy there is intense *pain in the epigastrium*. Gueneau de Mussy¹ regards a pain along the tenth rib, extending from

¹ Arch. gén. de Méd., 1853, vol. xi. Quoted by Fox.

PLATE XXVIII.

FIG. 1.—Anterior Aspect.

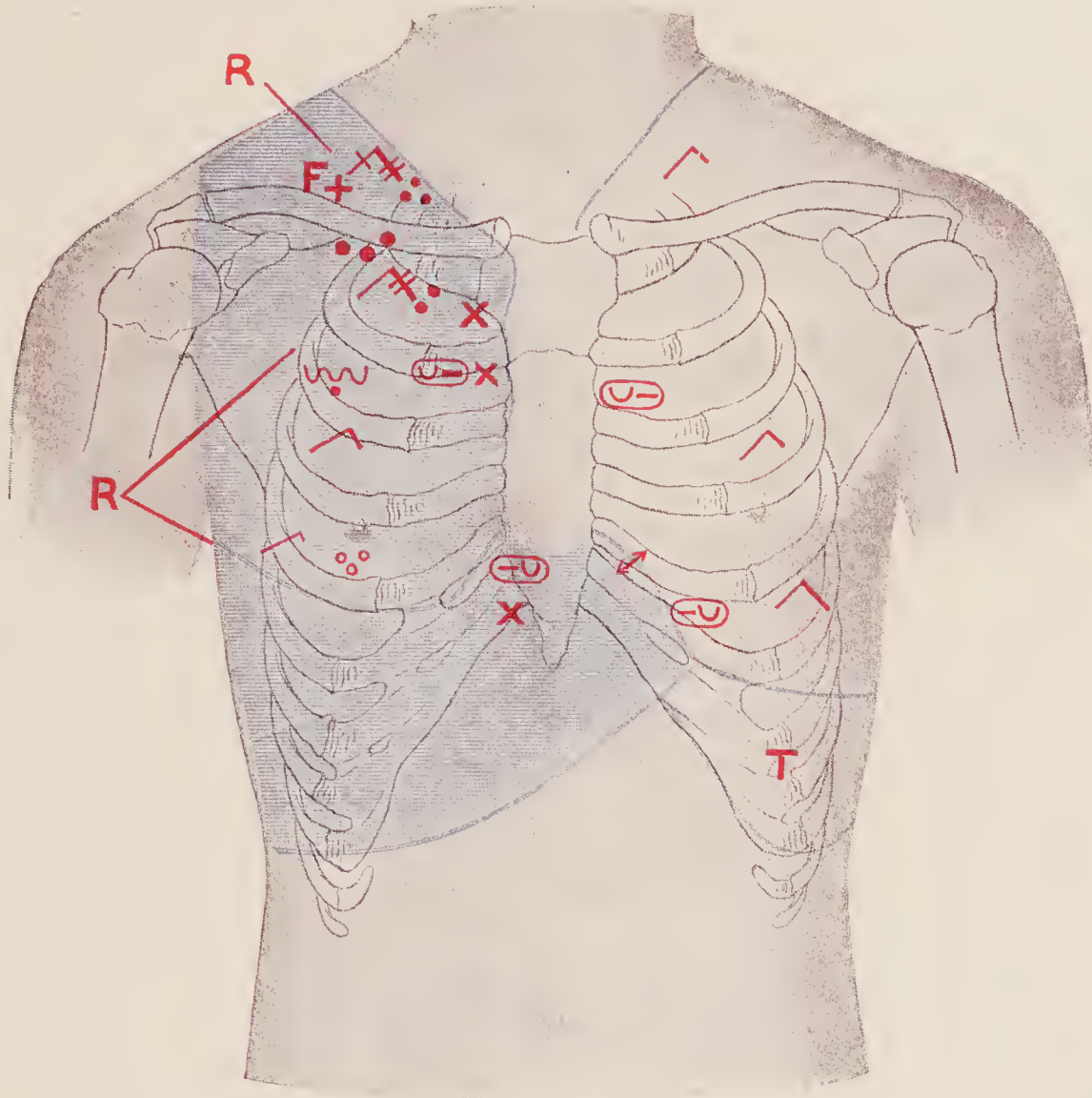
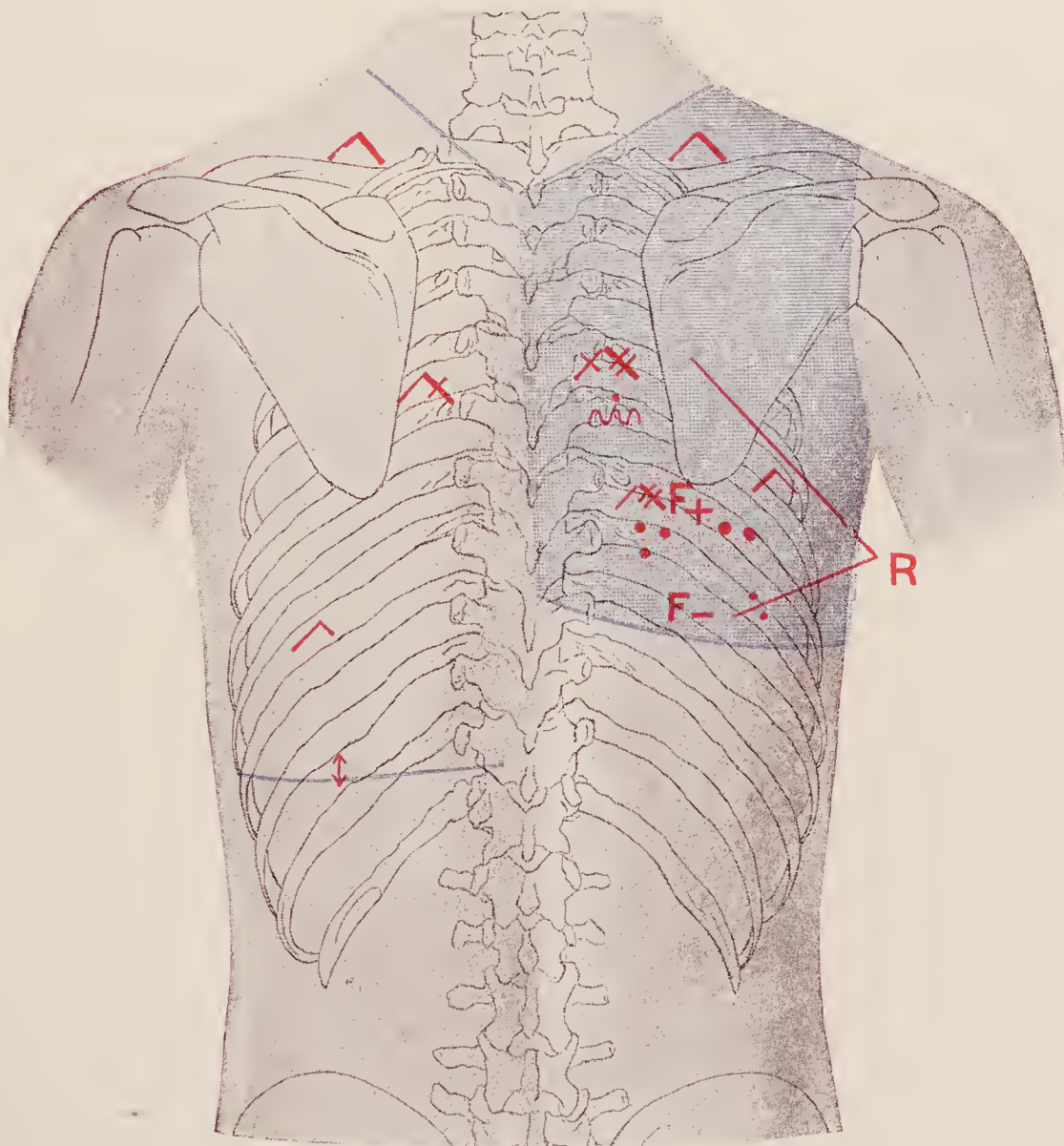


FIG. 2.—Posterior Aspect.



Fibroid Phthisis with Chronic Pleurisy.

Heart drawn toward the right and aorta uncovered by retraction of lung margin. Vicarious emphysema of left lung.

the anterior extremity to the sternum and xiphoid cartilage, as pathognomonic. Other symptoms are *nausea*, *vomiting*, and *hiccough*. The *dyspnœa* often amounts to orthopnœa, or the patient sits stooping forward. The anxiety of the patient is very great. The *fever* is usually higher than in ordinary pleurisy, and there may be delirium. Effusion may lessen the pain. Peritonitis may occur at the same time or be secondary to the pleurisy.

Diagnostic Features of Acute Pleurisy. The special features of diagnostic importance that are observed in the course of pleurisy are the subjective symptoms of pain, dyspnœa, cough, and fever, the physical signs of effusion within the pleura, and the results of exploratory puncture.

Symptoms. *Pain*: The pain is short, sharp, lancinating, and is usually recognized quite readily by its character and location. It must be distinguished from the pain due to pleurodynia and intercostal neuralgia. The pain of pleurisy is associated with cough and is increased by breathing. It causes diminution of movement of the affected side. The patient is compelled to sit up in bed, or lie on the side which is the seat of pain.

Cough: In the first stage the cough is short, suppressed, dry, and painful. It is constant. In the second stage it changes in character. There is no pain, there is no expectoration. It is frequent and irritating, and of a peculiar sound which is difficult to describe, and yet, when once heard, is most suggestive in subsequent cases. It is short and lacks resonant quality, as if the fluid in the chest stopped the sound-waves.

Dyspnœa in the first stage is due to pain, in the second stage to the large effusion which encroaches upon the normal air-space. It is not diagnostic.

Physical Signs. The most decisive signs of pleural effusion are *diminution or absence of movement*, *enlargement of the affected side*, *absence of fremitus*, *flatness on percussion*, *fulness of intercostal spaces*, and the *displacement of organs*. The latter is of the greatest diagnostic importance in the distinction between consolidation and effusions. The results of *exploratory puncture* lead to positive conclusions usually, although it must not be forgotten that effusions may be loculated, and therefore missed by the aspirating-needle. Again, the enormously thickened pleura may intervene between the exudate and the surface of the chest, and prevent withdrawal of the fluid. Finally, effusions may complicate inflammatory processes, as pneumonia, tuberculosis, or abscess of the lungs. Securing fluid for diagnosis by aspiration, therefore, does not necessarily exclude these conditions, and hence, before the process is decided to be within the pleura alone, the sputum and other conditions must be taken into consideration.

Differential Diagnosis. Acute plastic pleurisy is diagnosticated from *acute pneumonia* by the friction-sound and the maintenance of the clear percussion-note and the normal respiratory murmur, with unaltered vocal resonance and fremitus. When effusion takes place, the chest is enlarged and immobile, especially on the affected side; the interspaces are filled out and the diaphragm is depressed; these changes do not occur in pneumonia. Moreover, the percussion-note in pleural effusion is flat, with greatly increased resistance; the shape of the upper line of dulness is

diagnostic; the respiratory murmur is feeble and distant, or entirely absent, except along the spine, where the compressed lung yields bronchial breathing, and also above the line of effusion, where the lung yields exaggerated breathing. In pneumonia, on the other hand, the percussion-note is dull, without greatly increased resistance, and the breath-sounds are bronchial. In addition, in pleurisy, the vocal resonance and fremitus are usually almost if not entirely absent, and posteriorly at the level of the effusion ægophony may be detected. In pneumonia, on the contrary, vocal resonance and fremitus are increased in intensity. In pleurisy with effusion the movable organs are dislocated and Traube's line is obliterated.

Finally, the fever of pneumonia is much higher and more continuous than that of pleurisy, the respirations are more frequent, the cough is looser, and in typical cases is followed by rusty sputa. (Compare the temperature chart in article on Pneumonia.) A crucial test is aspiration with a hypodermatic needle; in pleural effusion, serum is withdrawn; in pneumonia, a few drops of thick blood.

In *pleurodynia* there is also severe pain in one side; but the pain is more continuous than that of pleurisy, and consists of a constant aching or a burning sensation. It is made worse by twisting or turning, as well as by breathing. The side is also tender to the touch. The pain is not so sharply localized as that of pleurisy, and may leave one side and affect the other. It is unaccompanied by fever or friction-sound, and is frequently found in rheumatic subjects.

In *intercostal neuralgia* there is the same absence of fever and friction-sound. The pain, however, is sharply localized, as in pleurisy, but is of the darting, neuralgic character, and is associated with tenderness at the points of exit of the intercostal nerves. It is most common in women, especially in those having uterine disturbances. It is more frequent on the left side, and just beneath the mammary gland.

Chronic Pleurisy.

Chronic dry, or plastic, pleurisy is the result of an acute attack, or develops insidiously if tuberculous.

Physical Signs. Chronic pleurisy causes great *deformity of the chest* from contraction, and *compensatory emphysema* of the healthy lung. The *heart is dislocated* or can not be found on physical examination, because it is overlapped by lung or is drawn behind the sternum. There is considerable *spinal curvature*, *dislocation of the scapula*, *deformity of the shoulder*, and indrawing and overlapping of the ribs at the base of the chest.

Chronic Pleurisy with Effusion. This results from an acute attack of pleurisy, in which the fluid remains unabsorbed, or from a series of attacks.

Symptoms. So far as subjective symptoms go it may remain latent; patients so affected not infrequently go about their work with comparatively little dyspnoea. There may be an evening rise of temperature and acceleration of the pulse. Chronic effusions are more likely to be purulent in children than in adults. When empyema results, the fever

PLATE XXIX.

FIG. 1.—Anterior Aspect.

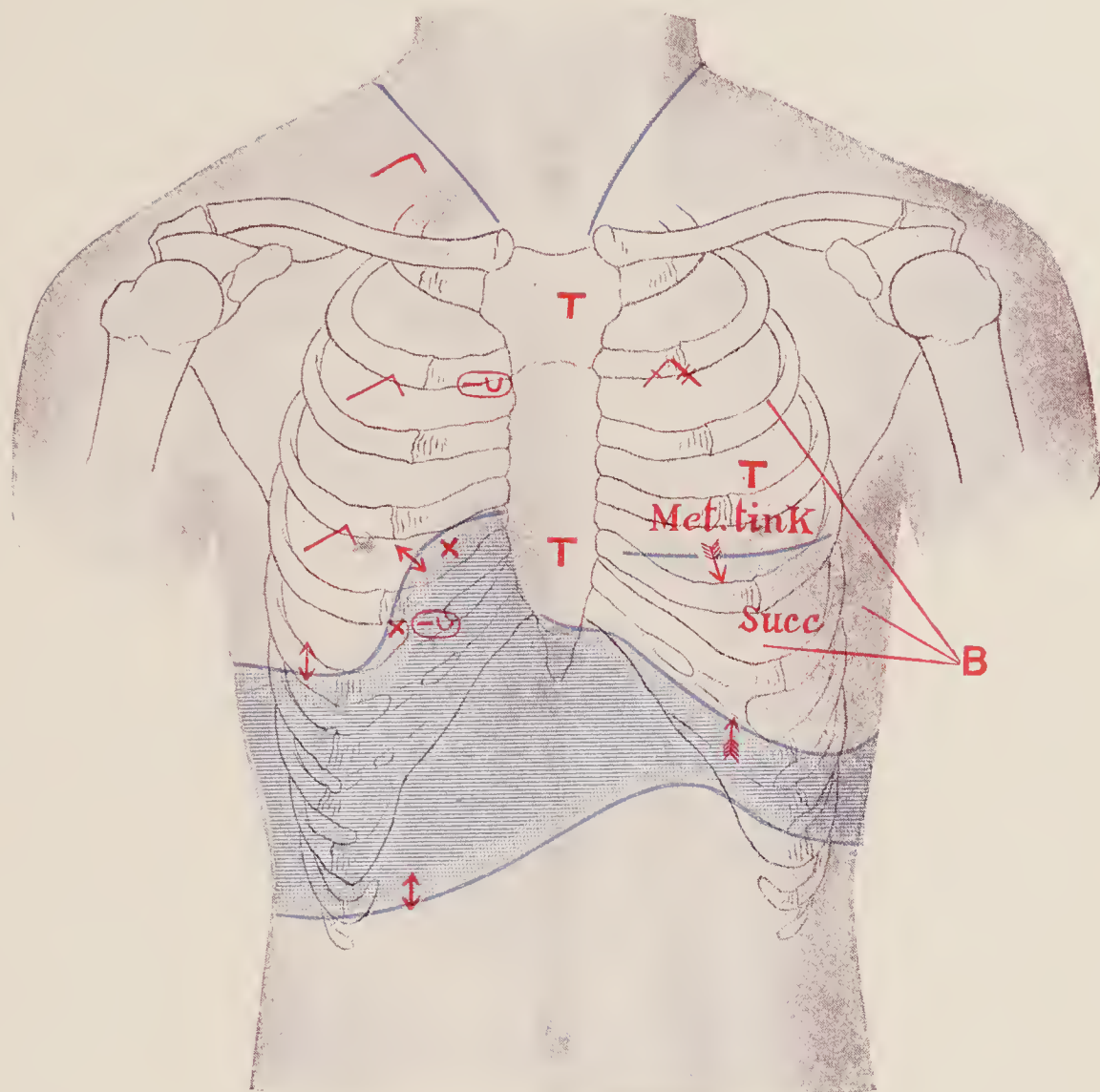
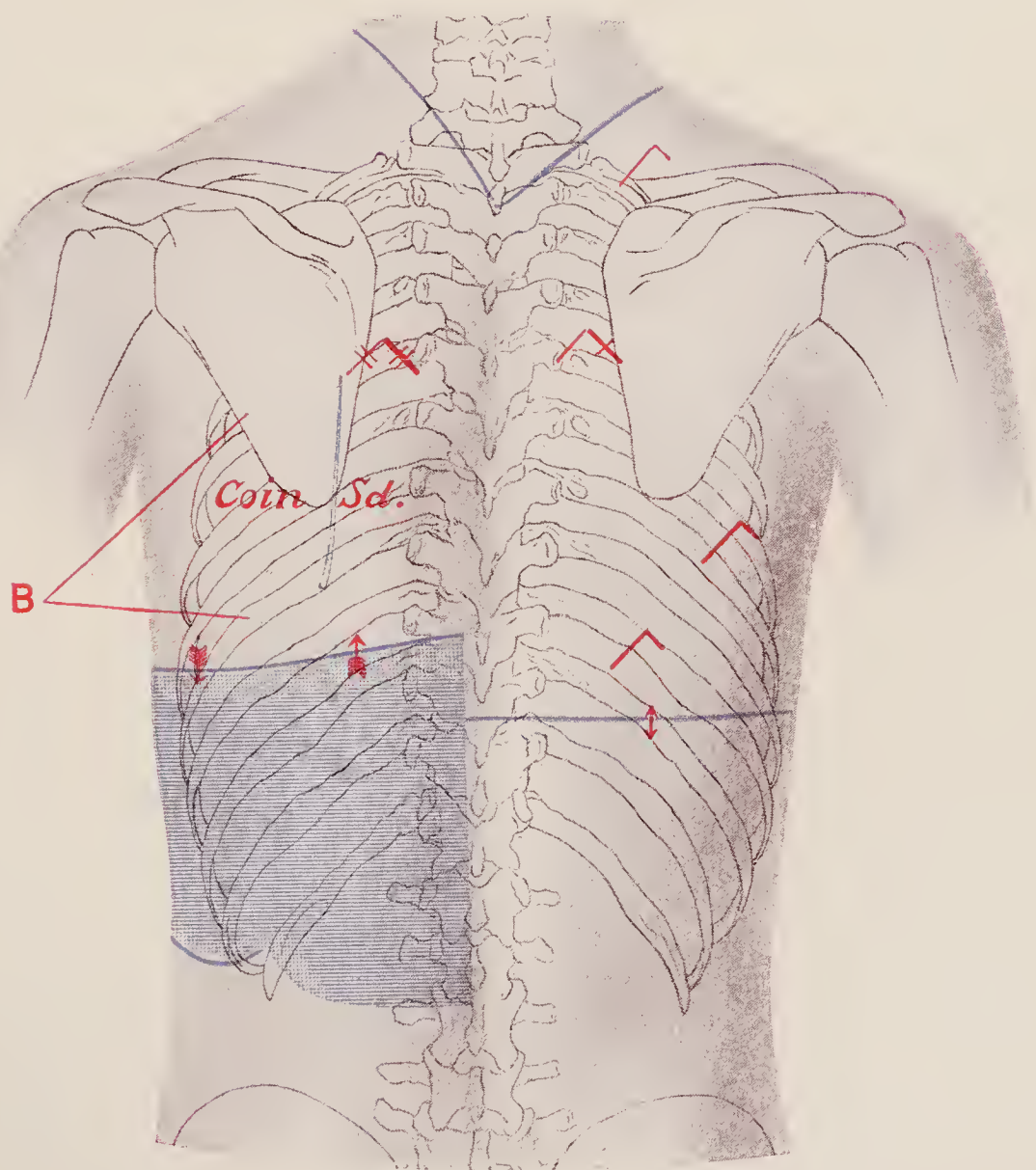


FIG. 2.—Posterior Aspect.



Pneumothorax (left-sided).

becomes hectic ; there are chills and sweats, pyæmia develops, and death is likely to occur from some intercurrent suppuration, as cerebral abscess.

Physical Signs. The physical signs are the same as in acute effusion.

After chronic effusion the chest is rarely restored to its original shape, even if the effusion is finally absorbed. The affected side becomes motionless and retracted. In course of time the spine may be bent. The opposite lung becomes hypertrophied. The patient is usually in precarious health, liable to acute attacks of pain in the affected side, and liable also to be carried off by phthisis or some intercurrent affection. Rarely the patient may maintain good health ; complete cure is even possible, with restoration of the retracted side to, or almost to, normal dimensions, especially in children.

Pneumothorax.

Pneumothorax consists in an accumulation of air in the pleural cavity, accompanied or followed by an outpouring of fluid, which may be serous or purulent, constituting respectively *hydropneumothorax* and *pyopneumothorax*.

Pneumothorax may originate : (1) in causes external to the chest, by perforation of the chest-wall and pleura ; (2) in perforation of the lungs, bronchi, or œsophagus ; (3) it may be caused by gases developed from an existing effusion.

The most frequent cause is tuberculous disease of the lung, and next, an empyema ; out of 121 cases collected by Saussier, 81 were due to phthisis and 29 to empyema. Pneumothorax may occur very early in tuberculosis of the lung, and may even be the first symptom of that disease. (See cases referred to by Fox and recorded by Louis and Chomel.) The left side is affected not quite twice as often as the right ; the disease is usually unilateral.

Symptoms. The onset of the condition is usually sudden. During a paroxysm of coughing or vomiting, or without immediate cause, there is an escape of air into the pleura, and in the majority of cases the patient at once complains of *acute pain in the chest* and *excessive dyspnœa* with *great dread of impending suffocation*. The patient often sinks into *collapse* from shock, but sudden death is rare. If the escape of air into the pleura is gradual, there will be less pain and dyspnœa.

Physical Signs. (Plate XXIX.) The *chest is distended*, especially on the affected side ; the *percussion-note* is a *bell-like tympany* except when the distention is excessive and the air contained is under great tension, when the note is proportionately duller and higher in pitch ; the *diaphragm is depressed* and the *heart displaced* unless adhesions prevent it. In left pneumothorax it may beat on the right side, the whole mediastinum being pushed to the right ; in right pneumothorax the mediastinum may be pushed to the left nipple ; hence there is resonance over the normal cardiac region. The pitch of the percussion-note may be raised when the mouth is closed, and lowered when it is open (*Wintrich's change of note*), and a *cracked-pot sound* can be elicited in some cases, but this occurs only when the communication with the pleura remains open.

A valuable sign of pneumothorax is the *coin-test*, or, as Trousseau named it, the *bruit d'airain*. A silver coin is laid upon the chest and struck with another, while the auscultator applies the stethoscope opposite to the point struck or over any part of the side distended by air. The ringing coin-sound is reproduced with great intensity. It is pathognomonic, and the outlines of the cavity can be traced by it.

When fluid is present, as it usually is, there will be the ordinary signs of a pleural effusion, which have been sufficiently dwelt upon. The fluid is more mobile in pneumothorax, however, than in simple pleurisy, so that its level changes more quickly with change of posture of the patient, and *Hippocratic succussion* is readily obtained. This movable dulness is a very valuable sign—indeed, being almost pathognomonic.

The lung being compressed against the spine by the air, as it is by the fluid in pleurisy, the *breath-sounds* are *feeble* or *absent*, except over the root of the lung, where the breathing is bronchial. But if the lung is not completely collapsed, *amphoric breathing* may be heard, the air-chamber of the pleura acting as a consonance-box; it may be heard with both inspiration and expiration, or only with expiration.

Metallic tinkling is a sound believed to be due to the vibration of bubbling bronchial râles re-echoed through the air-chamber, or to drops of fluid falling from above upon the surface of the effusion. Re-echoing, with metallic quality, may also accompany the heart-sounds, and in cases in which the respiratory murmur is amphoric the vocal resonance is of the same character. *Vocal fremitus* is generally *absent*.

Differential Diagnosis. Pneumothorax is most likely to be confounded with (1) emphysema; (2) tuberculosis of the lungs with large cavities; (3) cases of pleural effusion in which the lung is markedly hyper-resonant above the upper level of the fluid; and (4) abscess below the diaphragm containing air (*pyopneumothorax subphrenicus*).

Emphysema can be distinguished by its slow onset, its relatively slight impairment of the general health, by the fact that it is bilateral, whereas pneumothorax is almost always unilateral, and by the existence of feeble breathing with greatly prolonged expiration. Amphoric breathing and resonance, metallic tinkling, and signs of fluid are all absent in emphysema.

When the pneumothorax is circumscribed, the physical signs resemble those of *pulmonary cavity*. But over a large cavity the chest is usually flattened. Cracked-pot sound and alteration in pitch upon opening and closing the mouth are more common in cavity than in pneumothorax. Displacement of viscera does not necessarily occur in phthisical cavity, the coin-test is negative, and succussion can not be produced. Fremitus is absent in pneumothorax and increased over a cavity.

The hyper-resonance above a *pleural effusion* develops with a very different clinical history, is accompanied by increase of fremitus with bronchial or, at times, amphoric breathing, and changes when the patient's mouth is open or closed. The percussion-note usually lacks the metallic quality heard in pneumothorax, metallic tinkling is absent, the coin-test is negative.

Pneumothorax must be distinguished from abscess below the dia-

phragm containing air (*pyopneumothorax subphrenicus*). Often the distinction is difficult. The constitutional symptoms of suppuration are present. Leyden points out the importance of remembering the sequence of events in the development of the disease. When the abscess is situated below the diaphragm, abdominal symptoms precede its development, and early in the course of the disease there is absence of respiratory symptoms. If the patient has had gastric ulcer, this would point to subphrenic abscess, as most of the cases of subphrenic abscess are secondary to gastric ulcer. Moreover, in subphrenic abscess the heart is not displaced nor do the interspaces bulge. Indeed, the viscera below the diaphragm are more likely to be displaced than are those above it. In pneumothorax, according to Leyden, the respiration is normal under the clavicle, and the transitions from the normal to the metallic and amphoric sounds lower down are abrupt. In pyopneumothorax on the left side the semilunar space disappears. In subphrenic abscess the amphoric sounds laterally or posteriorly may be above and below the diaphragm, or they may be loudest at the epigastrium. In addition, in *pyopneumothorax subphrenicus*, as Mason points out, adhesions of the lung to the diaphragm and parietes can be made out, particularly if the case has been under observation in its earlier stages and dry pleurisy has been discovered. Abscess in this location and slight fluctuation are likely to develop with associated effusion. The limited extent of the effusion is of diagnostic import in favor of subdiaphragmatic inflammation.

CHAPTER VII.

DISEASES OF THE HEART, THE BLOODVESSELS, AND THE MEDIASTINUM.

DISEASES OF THE HEART.

Symptoms. The symptoms of disease of the heart are due to the anatomical structure of the organ, to its physiological offices, and to the morbid process. The heart is a hollow muscular structure which hangs in a cavity, the pericardial sac, and encloses other cavities—the two auricles and two ventricles—separated from each other by valves. Both sets of cavities are lined by serous membrane. The serous membranes are subject to similar lesions, and present similar signs as diseased serous membranes elsewhere. In inflammation of the external membrane—the pericardium—the surfaces rub together and create a sound of friction. The external serous cavity may also become filled with the products of exudation or transudation. Physical signs are produced. They are the physical signs of a localized increase of contents as determined by inspection, palpation, and percussion, and of physical interference with the heart's action. The heart muscle is also subject to the same morbid processes as other muscular structures. They are hypertrophy and atrophy; inflammation, acute and chronic, with over-growth of connective tissue; and degenerations. The *symptoms* are likewise the same. Increase or diminution in the power of the muscle is associated with corresponding change in size, which is determined by physical signs. Above all, however, such change modifies the heart's action so that strength or weakness of the muscle shows itself in excessive or deficient vascular pressure. The latter is more particularly an object of observation because of the congestions, dropsies, and cyanosis that ensue.

The heart is constantly subjected to internal pressure. Dilatation of the cavities or of a portion of a cavity (aneurism) follows previous disease of the muscle or increase of internal pressure, and causes physical signs of enlargement. Degeneration of the heart muscle, nearly always secondary to deficiency of vascular supply, is also attended by symptoms of weakness and physical signs of enlargement (dilatation) or of diminution in size (atrophy). When dilatation occurs, the orifices of the cavities enlarge, the valves can not close them, and the symptoms and physical signs of incompetency and of blood-regurgitation result.

The serous membrane that lines the cavities of the heart and, with the subserous tissues, makes up the structure of the valves, is subject to inflammations, the signs of which are common to all serous inflammations. The swellings and outgrowths that attend such inflammation occlude the orifices and prevent closing of the valves. There is produced

a physical interference with the heart's function which is recognizable by physical signs. The successful effort of the heart muscle to overcome such defects on the one hand (hypertrophy), or its failure on the other (dilatation), again leads to the production of symptoms and signs. The serous membranes, and hence the valves, are exposed to causes which excite inflammation. By virtue of the position of the heart at the centre of the circulation, the blood, infectious or irritative, as in rheumatism and Bright's disease, constantly bathes this vulnerable structure. For the same anatomical reason, symptoms arise not common to serous membrane inflammation—that is, *embolic* phenomena. (See Symptoms of Morbid Processes.) Hence, the physical signs (objective symptoms) of cardiac disease may be due to primary and secondary morbid anatomical changes. They may be due (1) to valvulitis as indicated by signs of (*a*) obstruction or regurgitation at the valve-orifice, or (*b*) of embolic phenomena; (2) to secondary changes in the heart muscle as seen in (*a*) change in the size and strength of the organ (hypertrophy or dilatation), and (*b*) in consequence of the latter, signs of congestion, œdema, cyanosis, etc.

It is the function of the heart to propel the blood. It has been shown how interference with the action of the heart muscle and with the consequent flow of blood through the cavities and orifices modifies this function. The functional power is increased or diminished by the physical changes. The evidence of increased power is increased force of the heart-beat, and increased pressure in the arteries (pulse).

Diminished power shows itself in symptoms of diminished blood-supply to parts, and in stagnation of the blood that is sent to the periphery. The former is more pronounced in cerebral anæmia, and physiological weakness of organs or of the organism as a whole; the latter, in congestion and dropsies.

The functional activity of the heart is controlled by a nervous mechanism, any alteration of which alters cardiac action and consequently produces symptoms. Just as with the larynx, a break in the cardiac mechanism may be in the centres in the medulla, in the centres in the heart muscle, or in the sympathetic nerves to and from the heart. The rich anastomosis of these nerves exposes the heart to disturbance by reflex influences. We should suppose such extensive innervation would invite frequent cardiac perturbation. In a measure it does, but, fortunately, so perfect is this mechanism that the inhibitory fibres control such perturbation to a large extent, and we do not see such pronounced symptoms as occur in disease of the larynx. The symptoms which point to disturbance of the cardiac mechanism are alterations in the rhythm of the heart. Its action may on this account be increased or diminished in frequency, or it may be irregular or intermittent. Such alterations of rhythm may be due to organic disease of the centres, notably the pneumogastric from apoplexy, softening or tumor in the medulla, or to stimulation or depression of the centres by toxic substances in the blood, as in anæmia, acetonæmia, or autogenetic or other toxæmias, or by nicotine or other extraneous material. The altered rhythm may be, and most frequently is, of reflex origin. It may be due to disease of the nerves, as the pneumogastric or sympathetic, from pressure upon the nerve-trunk

by a tumor or inflammatory growth. The most pronounced symptom of altered rhythm of which the patient is cognizant is palpitation. The exciting cause of this, as well as of other rhythmical changes, must, in the great majority of cases, be sought for beyond the domain of the heart.

While the symptoms or signs of cardiac disease are often due to morbid processes in the heart or its membranes, it must be remembered that grave and persistent subjective and objective symptoms may be caused by, or at least associated with, disease of contiguous structures outside of the pericardium. The symptoms are not excited through the nervous system, but are produced by mechanical encroachment upon the organ, as in pleurisy with effusion, mediastinal disease, and disease of sub-diaphragmatic viscera. They will be referred to in the study of objective symptoms. Care must be taken never to overlook the possibility of their presence.

In the study of the symptomatology of cardiac disease the student must bear in mind two things: (1) that the cause of the morbid processes and of the symptoms (pain and palpitation) may be elsewhere than in the heart; and (2) that the ultimate object of the examination is to determine the muscular power of the heart. He will soon learn that with that power intact the functions can be performed notwithstanding the presence of marked physical abnormalities.

The recognition of disease of the heart is usually not attended by much difficulty except in some special lesions. The non-recognition of cardiac disease is due to faults in the examination. The physician is too often satisfied with the recognition of the remote process, as a congestion or functional weakness in some organ. Safety lies, as has been often said, in the examination of the organs of the body. Often, for instance, indigestion from gastric catarrh is not relieved, for the cause, mitral regurgitation, is not recognized.

The Data Obtained by Inquiry.

Diagnosis. The historical diagnosis furnishes facts of ætiology which clear up doubts about an obscure case. Excesses of all kinds, infections, and intoxications, are causal, and hence diagnostic.

The subjective symptoms in diseases of the heart and bloodvessels are common to many affections and most of them are considered in the section on Subjective Diagnosis. They are pain, palpitation, dyspnœa, cough, arrhythmia, and pulsation of the vessels. They are not so frequently found in other affections, but are sufficiently common to be treated of in a separate chapter.

The subjective symptoms therefore are: (a) Cardiovascular—pain, palpitation, arrhythmia, and pulsation. (b) Pulmonary—dyspnœa and cough. (c) Gastro-intestinal—the symptoms of gastritis and enteritis. (d) Laryngeal—pain, aphonia, and cough. (e) Renal symptoms of congestion of the kidney, of chronic inflammation (cyanotic induration), and renal embolism. (f) Hepatic—pain, fulness, and pulsation of the liver. (g) Nervous—vertigo, chorea, convulsions, coma, and disorder of special senses.

The physical diagnosis establishes or renders final the conclusions as to the nature of a cardiac lesion.

The laboratory diagnosis enables us to discover the effects of cardiac disease and practically establishes a diagnosis of infectious endocarditis.

Pericarditis.

Inflammation of the Pericardium. The inflammation may be *acute* or *chronic*. It is also divided according to the nature of the inflammation into *simple fibrinous* inflammation and inflammation with *effusion*. The effusion may be serous (really serofibrinous), bloody, or purulent, depending upon the nature of the inflammation. Pericarditis may be *primary* or *secondary*. The primary form is of extremely rare occurrence. Indeed, it may well be doubted whether, in common with the inflammations of serous membranes in general, pericarditis is ever primary, or so-called idiopathic, in origin.

Causes. 1. EXTENSION FROM NEIGHBORING STRUCTURES. Extension of the inflammation from infected tissues in the vicinity is a common cause of pericarditis. It may follow a pleurisy and partake of the nature of the primary pleural inflammation. It often attends empyema, either from extension of the infection to the pericardium or from rupture into the pericardial sac. It may follow all forms of inflammation of the mediastinum, and sometimes results from extension of infection from a tuberculous peribronchial gland. Disease of the ribs adjacent to the pericardium may set up pericarditis, acute or chronic. It attends aortic aneurism at times, but more frequently infectious endocarditis and myocarditis. Inflammations below the diaphragm frequently give rise to pericarditis. Peritonitis, when general or local; subdiaphragmatic abscess; suppurative gastritis, with perforation of the stomach; abscess of the liver; suppurating hydatid and other forms of suppuration below the diaphragm belong to the latter.

2. GENERAL INFECTIONS.—The general diseases causing inflammation of the pericardium are those which affect serous membranes. They are: infectious diseases, particularly scarlet fever, measles, erysipelas, and typhoid fever. All forms of septicæmia may be attended by inflammation of the pericardium. Tuberculosis is a frequent cause of pericarditis. Inflammation of this membrane frequently arises in the course of rheumatism. It may occur in the course of the disease, or attend some of the affections which are themselves manifestations of rheumatism, such as acute tonsillitis. In the course of certain dyscrasias the pericardium is frequently the seat of inflammation because more vulnerable. This is particularly the case in scurvy. It occurs also in Bright's disease, and may be the first manifestation to the patient of this disease, particularly in the chronic form of nephritis. It occurs in the course of gout.

The various forms of pericarditis may occur at any age, although that which attends scarlatina and rheumatism occurs in early life, while late in life it is an attendant upon chronic Bright's disease and gout.

Acute Fibrinous or Plastic Pericarditis. This is the most common form. It attends Bright's disease, rheumatism, and tuberculosis. It may

be wanting entirely in symptoms. A routine examination of the heart may reveal its presence.

In the course of one of the primary causal diseases, if the temperature rises a little higher than it should, or convalescence is delayed, pericarditis should be suspected. Again, if the pulse is more rapid and quicker than is customary at the period of disease the examination is made, or out of proportion to the temperature, the disease should be suspected. There may be altered rhythm or tumultuous action. In other instances the patient may complain of pain in the region of the heart. It is usually localized in the fourth or fifth interspace. It is not very severe and not influenced by pressure. Sometimes the pain is complained of at the xiphoid cartilage. In rare instances it may resemble that of angina pectoris. The pain and the occurrence of fever further call attention to the heart.

Physical Diagnosis. **INSPECTION.** Nothing unusual is observed, although the heart may be seen to beat more violently against the chest-wall. The impulse is diffused.

PALPATION. A friction-fremitus may be detected, due to the rubbing together of the roughened pericardial surfaces. It is not always present. It may be felt when the whole hand is laid over the præcordia, or by palpation with the tips of the fingers. It is most marked over the right ventricle, particularly in the fourth interspace, and is increased when the patient leans forward.

AUSCULTATION. A friction-sound is usually present. It may be present while the fremitus is absent; but, on the other hand, if the fremitus is present, we can always hear the friction. It is heard over the region where the fremitus is felt.

Point of Maximum Intensity. It may be heard along the course of the sternum. It is usually heard in the third or fourth interspace, but may be heard as high as the second, adjacent to the sternum in either interspace. Sometimes it is heard at the second costal cartilage on the right, rarely at the apex. The point of maximum intensity may vary with the position of the patient.

Time. It is both systolic and diastolic. In some cases it may be only systolic in time, or it may be of a galloping nature, representing three sounds during the cardiac cycle. Again the to-and-fro sound is not synchronous with the systolic and diastolic sound, although it occurs but once in the cardiac cycle. It may begin after systole, and be completed before the end of the diastole. The impression that it is a superadded sound is most positive.

Direction of Transmission. It is localized, and not transmitted.

Character. A friction-sound is a to-and-fro rubbing, scratching, or grating noise; it gives the impression of being near the ear. It may be modified by the pressure of the stethoscope by the position of the patient, and by the respiratory movements, which may press the heart firmly against the chest. It may be heard in the erect and disappear in the recumbent posture.

Diagnosis. Acute pericarditis is overlooked because it is not sought for. In the larger number of cases there have been no indications of disease of the pericardium. The diagnosis is usually easy.

DIFFERENTIAL DIAGNOSIS. The *pericardial friction* may be mistaken for an *organic heart-murmur* or for *pleural* or *pleuropericardial friction*. It is often difficult to distinguish the to-and-fro friction from the murmurs of double aortic disease. If attention is paid to the general and local phenomena, the mistake is not likely to be made. The location of the murmurs in organic heart disease, the direction of the transmission, the character of the murmur, the peculiar character of the pulse, and the secondary effects upon the muscle of the heart, point to the valvular lesion.

The pleuropericardial friction which simulates pericardial friction usually occurs in the course of phthisis or pleuropneumonia. It is modified by respiratory movement: (1) it may disappear, or at least diminish, if the breath is held; (2) a full expiration may cause its disappearance. While it is of cardiac rhythm, it is modified by the respiratory rhythm, so that on inspiration it is usually more marked. The pleuropericardial friction is not so strikingly modified by position.

Pleural Friction. This is of respiratory rhythm and ceases with cessation of breathing. The pericardial friction persists even if the breath is held.

Pericarditis with Effusion. As with fibrinous so with this form, it is frequently overlooked. This is because it very often develops without symptoms. In plastic pericarditis we have referred to the occurrence of *pain*. This may occur before the effusion in the latter form, but is usually moderate. As with dry pericarditis, however, it may, in rare instances, be very severe, anginose in character, and be increased by pressure over the heart or on the pit of the stomach.

Symptoms. The symptoms are usually due to the special character of the inflammation and the presence of fluid in the pericardium.

1. **GENERAL SYMPTOMS.** In non-suppurative cases the symptoms are usually cerebral. Delirium may be moderate or maniacal. It must not be confounded with the delirium which occurs in the course of acute rheumatism with hyperpyrexia. Choreiform movements have been described in many cases. They may, however, be of rheumatic origin. Other cerebral symptoms, as hemiplegia and convulsive attacks, occur in the course of pericarditis, probably due to an associated endocarditis, causing embolism. In some cases albuminuria is found.

The general symptoms of pericardial effusion depend upon the nature of the primary disease and the character of the fluid. In *tuberculous pericarditis*, emaciation, irregular fever, sweats, and prostration ensue. In *purulent pericarditis* there may be recurring chills with a temperature-range decidedly intermitting, along with other phenomena of purulent accumulation. In a case seen in 1895 the patient was extremely debilitated and prostrated on account of pneumonia following influenza. He was extremely anæmic, and the blood-count showed diminution of red cells to one-half without other change. Every fourth day after a chill, the temperature would rise to 103° or 104° F. A friction-sound was detected after the second chill. It disappeared, but the physical signs of effusion were not positive. From the first the heart's action was so weak that the sounds were scarcely discernible. At the autopsy, 4 or 5 ounces

of pus were found in the pericardial sac. The purulent accumulation was the only lesion to account for the symptoms, and, we would say now, was no doubt a pneumococcus infection.

2. LOCAL SYMPTOMS. The local symptoms are due to the accumulation of fluid within the pericardium. *Dyspnœa* is the most common. The degree depends upon the amount of effusion. If the latter is large, there may be extreme orthopnœa; if the effusion is present for a considerable time, it may give rise to no symptoms. *Dysphagia* may occur in large effusions on account of pressure upon the œsophagus. *Altered cardiac rhythm*. The effect of the effusion upon the heart is to interfere with its action. Although usually regular, on the slightest exertion or the least excitement it palpitates violently or becomes irregular. The heart's action is increased in frequency; when the effusion is very large, it may be not only irregular, but also intermittent. *Aphonia* may occur from pressure upon the recurrent laryngeal nerve. *Cough* of an irritative character is sometimes noted. The *pulsus paradoxus* may be present.

Physical Diagnosis. (Plate XXX., Fig. 1.) **INSPECTION.** There is bulging of the præcordia, particularly in children. The ribs and interspaces are prominent. In adults the interspaces are on a level with or distended beyond the surface of the ribs, and are sometimes widened. The enlargement may extend to the anterolateral region of the left chest. A large effusion interferes with expansion of the lung on the left side, and hence movement is diminished. The epigastrium may be prominent, on account of displacement downward of the diaphragm and liver. The apex-beat is absent or faintly seen, displaced upward and to the left. It does not extend as near the left border of dulness as in dilatation. It may be seen in the fourth interspace, or a faint impulse may be observed in the second and third interspaces beyond the midclavicular line.

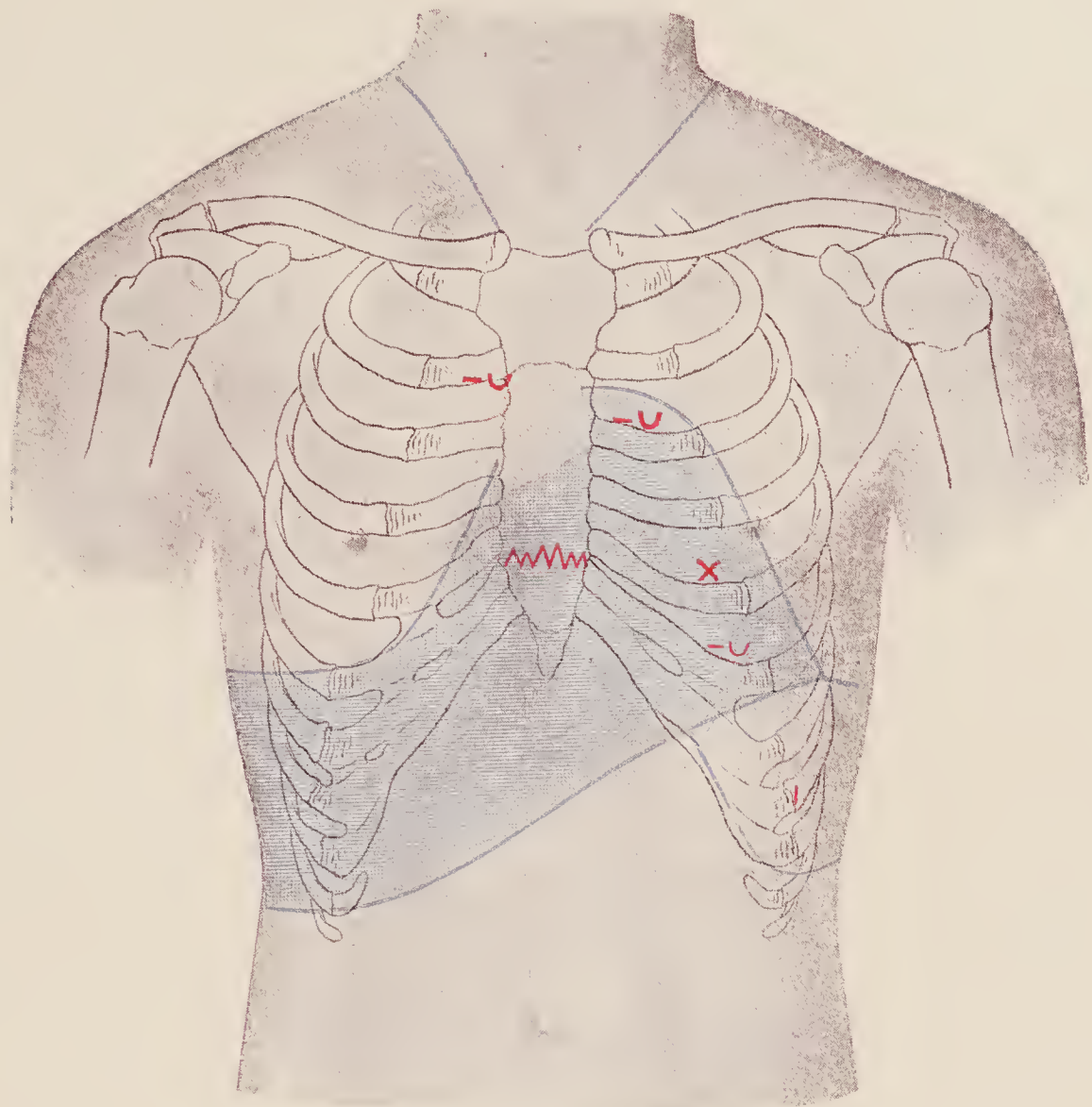
PALPATION. The impulse is feeble and diminishes in force as the effusion increases. The position of the apex as determined by inspection is confirmed. Ewart points out that the first rib is palpable at its sternal attachment in pericardial effusion. The pericardial friction which may have been present at first disappears with the effusion. Fluctuation may be detected in large effusions. The liver in large effusions is depressed and readily palpable.

PERCUSSION. The area of præcordial dulness is increased. There is increase of the lateral boundaries and great increase of *absolute dulness*. The relative dulness is generally absent. The increase of dulness is usually in all directions, although increase upward and to the left only is very common. It may extend as high as the second rib. As pointed out by Rotch, dulness in the fifth right interspace in the angle formed by the right border of the heart and the right lobe of the liver is common in effusion. It may be an early sign. Ebstein calls this region the *cardio-hepatic triangle*, and points out that the dulness is absolute in effusion, although impaired in normal states because of proximity to the liver.

Pulmonary resonance is modified posteriorly in large effusions. The dulness in large effusion includes the axillary region, so that it may simulate pneumonia or pleural effusion. The dulness, however, does not

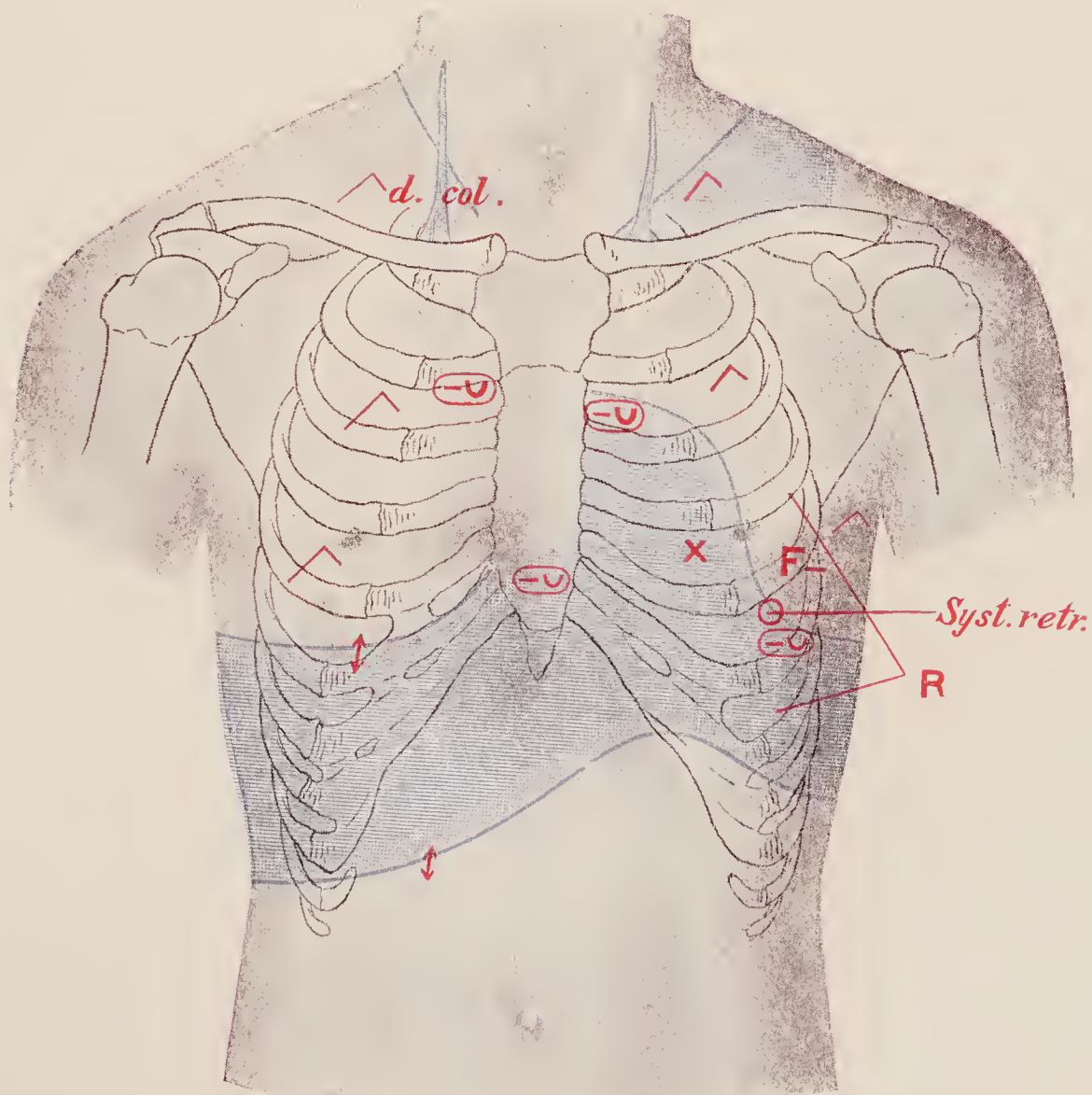
PLATE XXX.

FIG. 1.



Pericarditis with Effusion.

FIG. 2.



Adherent Pericardium. Chronic Left-sided Pleurisy.

extend below the eighth rib in this region, whereas in pleural effusion dulness always extends to the bottom of the pleural sac. In a large pericardial effusion the semilunar space of Traube is obliterated.

AUSCULTATION. The heart-sounds are feeble and distant. They may be scarcely heard at all over the præcordial region. The sounds at the base of the heart are diminished in intensity. If a friction-sound was heard at the beginning, it disappears entirely as the effusion is poured out. In moderate effusions the friction may be heard when the erect posture is assumed.

It must not be forgotten that the physical signs, and especially the change in impulse and the area of præcordial dulness, are modified by the position of the effusion. Accumulations occur behind the heart or above it, and in these situations interfere least with the displacement or the enfeeblement of the apex-beat. The area of dulness, however, is increased upward.

In cases of large effusion the compression of the lung may cause bronchial breathing heard posteriorly or in the axillary region. In a case under my care the diagnosis of pericardial effusion was readily made, but the enormous effusion so markedly simulated an effusion into the pleural cavity that both serous cavities were believed to contain fluid. Aspiration was performed in the sixth interspace in the anterior axillary line. The fluid was removed from the pericardium, as was afterward determined. During life pressure-signs—laryngeal stridor, difficulty of deglutition, and extreme dyspnœa—were present. Early vomiting, epigastric pain and tenderness, slight delirium, albuminuria, and an excessively weak, rapid pulse occurred in the course of the disease. The patient was a male, twenty years of age. The effusion was due to tuberculous pericarditis, secondary to tuberculosis of the bronchial glands. The physical signs were prominence of the præcordia; bulging of the interspaces on the left side; diminished expansion of the left side—anteriorly, laterally, and posteriorly; increased expansion at the extreme apex of the lung. The vocal fremitus was absent below the second interspace in front, below the third in the axilla, and diminished below the spine of the scapula behind. There was dulness from the second left rib in front to the margin of the thorax; from the fourth to the eighth rib in the axilla; below the eighth rib, tympany. The dulness extended beyond the margin of the sternum on the right side, almost to the right nipple-line, in the fourth and fifth interspaces. Posteriorly, dulness from the middle of the scapula to the base of the thorax, except along the vertebræ, where, from the seventh to the ninth rib, there was tympany. On auscultation the physical signs of pericardial effusion were marked. In the axilla the breath-sounds were absent. There were bronchial breathing and bronchophony behind from the spine of the scapula to the base along the vertebræ. They were most marked opposite the angle of the scapula, where the above-noted tympany was observed. In the midscapular line the breath-sounds diminished from above downward, and were absent at the base. It is seen that the physical signs of pleural effusion were present posteriorly and laterally, due to the enormous effusion. At the autopsy the pericardium was found to contain 64 ounces of fluid.

Pleural effusions may be excluded in similar cases by the absence of

dulness in the axillary region below the eighth rib ; by increase in dulness beyond the right edge of the sternum ; and at the same time by the absence of signs indicating dislocation of the heart to the right.

Diagnosis. Pericardial effusion must be distinguished from *dilatation* of the heart. Although feeble and diffuse, the expansile shock of the impulse is more distinct than in dilatation. This distinction is generally not difficult if the patient has been under observation during the development of the disease. The impulse is not always absent in dilatation. Fluctuation may be detected. The area of dulness in dilatation does not extend upward except in cases in which the right auricle is enlarged. The dulness does not extend downward in dilatation without a similar displacement of the apex-impulse. The shape of the dulness differs. In dilatation the dulness is square in shape ; in effusion it is triangular or pear-shaped, with the base downward. Normally, and in case of enlargement of the heart, the cardiohepatic angle is a right angle, sometimes even an acute angle ; in pericardial effusion this angle becomes markedly obtuse. In dilatation the sounds are accentuated, and are of a valvular character ; in effusion they are muffled. Dilatation does not cause the pressure-symptoms that occur in effusion. In pericardial effusion Bamberger's sign is of importance. When the patient is sitting upright, an area of dulness about the size of a silver dollar can be marked out at the angle of the scapula. Over it, dulness, increased fremitus, and bronchial breathing are made out. If the patient leans forward, the dulness and the other signs of consolidation disappear, to return when he sits upright. In children, pseudopleuritic signs are often present posteriorly—dulness, pleuritic friction, broncho-ægophony—but will disappear if the patient is put in the knee-chest posture. It is of diagnostic significance to have change of the rhythm and the character of the sound from day to day, or of its degree of loudness on movement of the patient.

The friction-sound may return after absorption of the effusion. It may disappear entirely and all signs of pericardial inflammation subside. Adhesion of the two layers of the pericardium may take place in plastic pericarditis and pericarditis with effusion.

Effusions into the pericardial sac of serum, blood, or air may take place without previous inflammation.

Hydropericardium. This may occur in the course of general dropsy from kidney or heart disease. It may not prove fatal of itself, but when associated with effusion in the pleural sac it contributes to the orthopnoea, which may cause death. Rarely after scarlet fever, effusion into the pericardial sac may be the only dropsical symptom. The physical signs are those of effusion. It is not attended by fever. It is frequently overlooked, because investigation beyond the pleura is not made after an effusion into that cavity has been found.

Hæmopericardium. This occurs on account of rupture of an aneurism of the first part of the aorta, of the heart itself, or of the coronary arteries. Wounds of the pericardium and heart cause hæmopericardium. The extension of the ulceration of malignant endocarditis to the surface may cause gradual effusion of blood.¹ The physical signs are those of

¹ See Keating, Transactions of the Philadelphia Pathological Society, 1878.

effusion. Death usually takes place before there has been time to make a sufficiently accurate examination to determine its presence. Rapid heart-failure due to compression is the cause of death. In the case referred to above, and in cases of rupture of the heart, the patient may live for many hours with dyspnœa and progressive weakening of the heart. In tuberculosis and cancer the effusion is frequently blood-stained.

Pneumopericardium. This occurs very rarely, and is due to perforation from without by a stab-wound, or perforation from the lung, œsophagus, or stomach. A purulent exudation may undergo decomposition, causing an accumulation of gas. If it arises from perforation, acute pericarditis is set up. The accumulation of gas causes tympany over the movable area of percussion-dulness. The most striking sign is noted on auscultation. Churning, splashing, or metallic sounds are heard, drowning the feeble heart-sounds. Death usually occurs quickly.

Adherent Pericardium. (Plate XXX., Fig. 2.) Chronic adhesive pericarditis may follow the acute form or, particularly if tuberculous, it may develop independently and progress slowly.

Inspection and Palpation. Indrawing of the interspaces may be seen at the time of the systole of the ventricles; even the ribs are said to be drawn in. This indrawing is most marked at the apex, and must not be confounded with the retraction that occurs in the third and fourth interspaces with the ventricular systole. The recession is synchronous with the systolic shock. In some cases the systolic movement over the præcordia is of an undulatory character. Walter Broadbent calls attention to systolic retraction of the left back in the region of the eleventh or twelfth rib as a valuable sign. The apex is displaced outward and the area of impulse is increased. The increase in area of impulse is due to the hypertrophy which always attends universal adhesion of the pericardium. After the systole there is frequently felt a quick rebound, known as the diastolic shock, which is said to be characteristic of pericardial adhesions.

In pericardial adhesions Friedreich's sign, *collapse of the cervical veins* during the diastole of the heart, is seen. We may also see inspiratory swelling. (Kussmaul.) In addition, the *pulsus paradoxus* is significant of the presence of pericardial adhesions, or rather of the dilatation that succeeds the adhesions. The pulse is small and feeble during inspiration, assuming greater strength during the period of expiration.

Percussion. The area of cardiac dulness is increased usually upward, extending as high as the first interspace. The area of dulness is frequently not modified by respiration—that is, it is not lessened when the patient takes a full breath, when the lungs should expand over the præcordial region. This is particularly the case when there is pleuritis associated with pericarditis, a common association in the large majority of cases.

Auscultation. On auscultation the signs vary. The sounds are due to hypertrophy or to dilatation; and it must not be forgotten that they frequently arise on account of pericardial adhesions. In the former condition the first and second sounds are accentuated; in the latter, a murmur may be heard at the apex, loud and systolic in time.

In pericardial adhesions the physical signs depend upon the condition of the heart muscle at the time of the examination. At first we have the

physical signs of hypertrophy, with retraction of the interspaces, particularly at the apex, or the space at the xiphoid cartilage. This is particularly the case in young subjects. In the later period of the disease the physical signs of dilatation arise, indicated by increase in transverse dulness, enfeeblement of impulse and of sounds, with the development of a murmur at the apex, undulation of the veins in the neck, and the *pulsus paradoxus*. The physical signs of associated pleurisy aid in the recognition of adherent pericardium. Diminution of the breath-sounds, increase in the area of cardiac dulness, lessened fremitus in the neighborhood of the heart pointing to pleural thickening, serve as further evidence. Sansom considers the presence of pulmonary tuberculosis of value, as pointing to the occurrence of pericardial adhesions, for the associated pleural adhesions are likely to be attended by tuberculous pericarditis.

I have learned to suspect adhesive pericarditis in a young subject the victim of valvulitis, when the symptoms do not yield to treatment—in short, when the heart is not affected by digitalis. Unfortunately, the physical signs are often not conclusive.

The *subjective* symptoms of adherent pericardium are those of dilatation or hypertrophy of the heart, whichever is in excess.

Diagnosis. Briefly, fixation of the heart is the physical condition the signs of which are well summarized by A. O. J. Kelly as follows: In the diagnosis of adherent pericardium, most help will be derived from: a weak or absent apex-beat, especially in cases in which there is no increase in the area of cardiac dulness; systolic retraction of a considerable area about the apex; systolic retraction of the base of the left chest posteriorly; arrest of the normal respiratory movements in the epigastric angle; imperfect descent of the apex-beat during inspiration; inadequate or entire absence of shifting of the apex-beat, with change in the posture (lateral posture) of the patient; absence of change in the limits of the cardiac dulness during the respiratory phases; absence of increase of the cardiac dulness to the right, despite marked engorgement of the veins of the neck; a diastolic shock or rebound of the heart; evidences of dilatation or hypertrophy of the heart in the absence of valvular or other disease that might cause it; absence of the characteristic changes in the heart in the presence of definite valvular disease—that is, absence of the usual hypertrophy of the right ventricle in mitral disease and of the left ventricle in aortic disease; absence of pericardial effusion in the presence of pleural and peritoneal effusions; paradoxical pulse—inspiratory diminution in the force and volume of the pulse; diastolic collapse and inspiratory swelling of the veins of the neck.

In unusual cases the first evidence of adherent pericardium is ascites—a symptom-complex for which Pick has proposed the designation, “pericarditic pseudocirrhosis of the liver.” In reality, the condition is a manifestation of multiple inflammation of the serous membranes, “*multiple serositis*,” or *polyorromenitis*, of the Italians and others. The pericardium, pleuræ, and peritoneum (especially about the liver—Zuckergussleber) may be involved consecutively.

Multiple serositis, or chronic adhesive pericarditis, with ascites is recognized by attention to the following facts: a history of a previous attack

of acute pericarditis, pleuritis, or perihepatitis; the early occurrence and subsequent disappearance of the œdema of the legs; marked ascites, with little or no œdema of the legs; enlarged liver early in the course of the disease (in some cases the liver appears not to have been enlarged); small and distorted, but otherwise smooth liver in the later stages of the disease; absent or very late enlargement of the spleen; repeated attacks of pain, tenderness, rigidity, and possibly palpable and audible frictions in the right hypochondriac region—attributable to attacks of perihepatitis; rapid recurrence of the ascites after tapping, and the physical signs of adherent pericardium—without which, it may be said, the disease is incapable of diagnosis.

Obliterative pericarditis with ascites may be distinguished from cirrhosis of the liver: by the symptoms and signs of adherent pericardium; by the absence of the ætiological factors of cirrhosis of the liver; by the slow, insidious, protracted, and intermittent course of the disease; by the long periods of quiescence during which the ascites may remain stationary and the patient in good condition; by the entire absence or the transient presence of slight jaundice; by the absence of symptoms of portal congestion other than ascites; in some cases, by the association of an enlarged, smooth, and firm liver with marked ascites; and by the fact that in many cases the patient survives a large number of tapplings.

Indurative mediastinopericarditis with adhesion may occur with or without fibrous inflammation and adhesion of the structures in the anterior mediastinum. The pericardium is adherent and thickened. Rarely the anterior mediastinum alone is a mass of fibrous inflammation. Peritonitis and perihepatitis may be found. The entire process is usually tuberculous.

The *symptoms* are dyspnœa, venous engorgement, cyanosis, enlargement of the liver, ascites, and dropsy.

The *physical signs* are those of extreme cardiac dilatation; the *pulsus paradoxus*; collapsing jugular veins during diastole, due to the dragging upon the innominate veins and cava by the fibrous adhesions, or to stretching and narrowing of the aortic arch by these adhesions; or inspiratory swelling of the veins of the neck. A friction-sound, systolic in time, heard over the sternum, increased when the arm is held up—*mediastinal friction*, so called—has been described in this affection.

Endocarditis.

Endocarditis may be acute or chronic. In either form it is usually secondary. The acute form is divided into simple and so-called malignant, infectious, or mycotic endocarditis.

Simple Endocarditis. Acute endocarditis rarely occurs primarily. It usually occurs secondarily to general morbid processes. The pathological antecedents are acute rheumatism, tonsillitis, whooping-cough, scarlet fever, gonorrhœa, rarely smallpox and typhoid fever. It is of common occurrence in pneumonia and tuberculosis. It is frequent in chorea. In the simple form it occurs in septic inflammations and in

debilitating diseases, as cancer. It may occur in gout and develop in the course of Bright's disease.

Symptoms. The symptoms of simple endocarditis are scarcely observed during the early course of the disease. The process is latent, and there are no indications of cardiac disease. The physical signs alone betray its presence. Unless these are sought for, the disease is overlooked. The subjective symptoms are negative. In the course of rheumatism or chorea, or during convalescence from the former, the patient may complain of palpitation, and increased frequency and irregularity of the heart. At the same time there may be a rise in temperature, not attended by any increase of the rheumatic symptoms; this should direct attention to the possibility of a cardiac complication.

The rise is not marked, and may not assert itself during the severity of the disease.

Physical Signs. On examination a murmur is detected in one of the cardiac areas. The murmur is soft, low in pitch, and follows the laws of transmission according to its situation. Instead of a distinct murmur a roughening of the first sound alone may be heard. Preceding the murmur the heart's action may be quickened and arrhythmical; the first sound may change in character from day to day or be accentuated; the second sound may be reduplicated at the apex and accentuated. The new sounds may disappear at first when the patient sits up; later they persist. The murmur must not be mistaken for the murmur at the apex in cardiac dilatation; or the murmur which may be heard in the course of fevers; or the murmur of anæmia, which may rapidly develop in rheumatism and other affections.

Malignant Endocarditis. Unlike simple endocarditis, the malignant form very rarely develops in the course of rheumatism and chorea. (See the Infections.) It occurs more frequently in pneumonia than in any other disease. It arises in the course of erysipelas, septicæmia, puerperal fever, and gonorrhœa. It may occur in dysentery. It is usually a streptococcus infection, but may be due to the staphylococcus, pneumococcus, gonococcus, typhoid bacillus, etc.

Symptoms. The symptoms are: (1) those due to the morbid process—the infection; (2) the physical signs; (3) those due to emboli. The *general symptoms* due to the specific morbid process are septic in nature. The febrile phenomena may be one of four groups: 1. The fever is paroxysmal. Chills and fever occur daily or at intervals of two or three days, resembling types of malarial fever. Each paroxysm is attended by profuse sweats. Rapid exhaustion ensues. The fever, instead of being distinctly intermittent, may be irregularly intermittent. 2. The fever is excessive and continued, and a typhoid state frequently sets in. The temperature is irregular; extreme prostration, low delirium, sordes, subsultus, and other symptoms of that state arise. 3. The fever is moderate and continued. Physical examination, however, reveals the presence of marked endocarditis. In this group chronic heart disease has usually preceded the affection. The duration may be prolonged. 4. The fever may be remittent. Petechial rashes and erythema are common, so that, as pointed out by Osler, the disease may resemble the eruptive

fevers. The *sweating* is profuse, contributing to the profound exhaustion which usually ensues. A septic *diarrhœa* occurs. In a few rapidly fatal cases *jaundice* has occurred. Again, the symptoms may be almost exclusively cerebral, resembling cerebrospinal or basilar meningitis.

The *embolic phenomena* are due to escape into the blood-current of soft vegetations from the valves of the left heart (for the right heart is rarely affected), which are carried by the blood-stream to distant points of the circulation. Emboli occur in the brain, producing convulsions, aphasia, or hemiplegia; they occur in the retina, causing some visual defect which may be accurately recognized by ophthalmoscopic examination. They occur in the lungs, producing local pain, hæmoptysis, possibly localized dulness, bronchial breathing, and râles. Emboli occur in the kidneys, producing bloody urine and renal pain. In nearly all cases the spleen is the seat of embolism, and in some instances infarctions may take place in this organ alone. The spleen is always enlarged, and the infarct may cause pain and increased tenderness on pressure. Emboli in the skin and mucous membranes present the most striking phenomena. The hemorrhages underneath the skin are minute. Emboli are seen in the extremities, but may also be found on the trunk. Occurring in the long bones, they cause local pain and tenderness. They occur in the mucous membranes, as those of the mouth and tongue. Emboli are seen in the bulbar conjunctivæ and in the conjunctivæ of the lids.

Physical Signs. Repeated examinations are necessary in some cases to determine the presence of a murmur, or to decide whether a previously existing organic lesion is the seat of an acute process. Variations in the character of the murmur from day to day are characteristic of malignant endocarditis. In organic *heart disease* with dilatation and failure of compensation, irregular fever followed by embolic phenomena points to the occurrence of an infectious process on the antecedent valvulitis.

Diagnosis. The diagnosis rests upon proof that an infection is present, and is made by the methods described in Chapter I., Part II., which should be reviewed by the reader. The history of an infection in some part of the body is most important in the diagnosis. The presence of the infection, as well as its nature, may be disclosed by an examination of the blood. When embolic phenomena are present, the diagnosis is made without much difficulty.

The more pronounced general symptoms distinguish it from *simple endocarditis*. The temperature range, the septic and typhoid symptoms, belong to the malignant form.

The more prolonged cases with moderately continuous fever, without apparent primary cause, are frequently confounded with *typhoid fever*. This is readily appreciated when the symptoms of the two are compared. The following symptoms are common to both malignant endocarditis and typhoid fever: a continued type of fever, with the symptoms of the typhoid state, including delirium; enlargement of the spleen, diarrhœa, and abdominal tenderness; there may be infarctions, although they are extremely rare in typhoid fever, and only occur late in the disease; progressive exhaustion. But in endocarditis the onset may be more abrupt, and prostration and anæmia may develop very early, and sweating may

be profuse. The fever does not present the regularity of type that is seen in the development of typhoid. In endocarditis there is more chest oppression and dyspnoea early in the disease than in typhoid fever. In endocarditis the source of the infection may be discovered in the genito-urinary organs, the lungs, the bones, etc. The diazo reaction is found in typhoid fever after the fifth day, but rarely, if ever, in endocarditis. A polynuclear leukocytosis is practically present in malignant endocarditis, while leukopenia is characteristic of typhoid. The results of bacteriological examination of the blood, urine, feces, petechiæ or rose-spots, and especially of *serum diagnosis*, distinguish the two affections. This ought to be of value in endocarditis, because the process is usually due to a staphylococcus or streptococcus infection. Either micro-organism may be found in any suppurations which may possibly be present, or in the blood. In a child recently seen by me in the relapse of an attack of typhoid fever, malignant endocarditis was thought to be present because of a loud and rough murmur at the pulmonary orifice. Fortunately the murmur was present in the apyretic period, and as the child was anæmic its exaggeration was ascribed to the fever.

Malignant endocarditis must be distinguished from *cerebrospinal fever* and from *smallpox* of the hemorrhagic type. We must rely on the local cardiac symptoms and physical signs, and the preponderance of these over the other symptoms. Of course, the prevalence of an epidemic and a history of exposure are of service in the distinction between the diseases. Examination of the blood excludes the forms of *malaria* which formerly were mistaken for endocarditis.

There is probably no disease which is more frequently overlooked. It has been mistaken for cancer of the stomach with secondary infection, tuberculosis, pernicious anæmia, Bright's disease with terminal infection, as well as the infections just considered.

Probably the local infection, which is causal and most frequently not sought for, is a latent gonococcus infection.

Chronic Endocarditis. Chronic endocarditis may follow the acute form or develop in the course of atheroma or of endarteritis due to alcoholism, the poison of syphilis or of gout. If associated with endarteritis, the endocardial change may be part of the general degenerative changes which occur in the aging process. It may be of dynamic origin, often following prolonged heavy muscular exertion, by which the valves, particularly at the aortic orifice, have been subjected to strain. The process is slow and insidious, and leads to the changes in the valve-segments which constitute chronic valvular disease.

Symptoms. The symptoms of chronic, or sclerotic, endocarditis are the symptoms of *chronic valvular disease*. *Insufficiency* or *obstruction*, or both combined, take place at the affected valve-orifice. The outflow of blood is retarded in obstruction. Backward flow, or regurgitation, takes place in the opposite direction from the normal blood-current in insufficiency.

When there is obstruction, *hypertrophy* usually develops to meet it.

Compensation. If the obstruction is moderate, and the person remains in good health, the hypertrophy is sufficient to overcome the obstruction.

In this manner the effect of the valve-lesion is compensated. On the other hand, when blood is permitted to flow by regurgitation backward into the cavity—that is, in the opposite direction to its usual course—it meets a blood-current flowing to this cavity in the normal direction, and the result is over-distention, or over-filling, of the cavity. Dilatation ensues, and may persist. If the regurgitation takes place suddenly, the dilatation continues; if gradually, as in chronic endocarditis, the dilatation is attended with hypertrophy. Thus, when there is regurgitation from the left ventricle into the left auricle, on account of incompetency at the mitral orifice, the auricle becomes over-distended with blood, for it is filling with blood from the pulmonary veins at the same time. This over-distention can only be overcome by some hypertrophy. When this is not sufficient, the blood is obstructed in the pulmonary circulation, with the consequences hereafter to be mentioned.

The symptoms of chronic endocarditis are latent if the lesions are compensated for, while by *physical diagnosis* alone are they recognized. The physical signs are those of *chronic valvulitis*. The character of the signs depends upon the lesion of the affected valve. If compensation is not established, symptoms of failure occur or *dilatation* of the heart arises.

Disease of the Coronary Arteries.

Atheroma, associated with the process in other vessels, or distinctly localized to the coronary arteries, affects these vessels. Its causal factors are those of endarteritis elsewhere. Its influence on the nutrition of the heart, either by sudden obstruction of the vessels by an embolus or by their gradual closure, with or without thrombus, is apparent.

Symptoms. If an atheromatous coronary artery is suddenly obstructed by an *embolus*, death may be instantaneous. This is a common cause of *sudden death*.

If a *thrombus* forms, it is followed by anæmic infarction, myocarditis, and mural aneurism with the usual symptoms.

The onset of symptoms may be sudden, although the process (occlusion) has been in progress for an indefinite time. (1) Præcordial oppression or dyspnœa or angina pectoris may be the first indication. (2) Succeeding this or immediately, dilatation of the heart, dyspnœa, and venous stasis occur. The heart's action is persistently rapid and may be arrhythmical. (3) The presence of an *aneurism* may be made out. If there has not been previous valvulitis, no murmurs are heard until dilatation ensues. The patient may live three or four weeks, or as many months.

In a large group of cases occlusion is so gradual as to lead to *myocarditis* (*q. v.*) only with the attending symptoms.

Diagnosis. Unfortunately, too often the diagnosis can only be provisional. Coronary artery disease may be suspected in sudden death if there has been a history of previous attacks of angina, if there is evidence of arterial disease elsewhere, and if dyspnœa or anginoid symptoms preceded the fatal termination.

Thrombosis secondary to atheroma may be suspected if a patient, in whom there is no valvular disease, no pulmonary or renal disease, is

seized with angina pectoris or dyspnœa; providing tachycardia and arrhythmia follow, and in a short time cardiac dilatation, venous stasis, etc. In a male, aged forty-three years, without syphilis, but with a history of antecedent rheumatism, an attack of angina pectoris followed some unusual exertion. Prior to this he had been in the most perfect health. The attack was followed by dyspnœa and remarkably rapid heart action without apparent cause. The physical signs of acute congestion of the lower lobe of the right lung followed within twenty-four hours of the attack of angina. The patient was ill three months. He improved somewhat, but rapidity of the heart's action and some stasis in the lung persisted. Gradually cardiac dilatation ensued, with a murmur in the tricuspid area. Death took place from pulmonary congestion. At the autopsy the coronary arteries were atheromatous; the left was filled with an old thrombus; there were extensive myocarditis and an aneurism of the left ventricle.

In another case, male, aged seventy-two years, with general atheroma but no valvulitis, sudden præcordial distress, tachycardia, and persistent dyspnœa were followed by cardiac dilatation, mitral incompetency, and general anasarca.

I have said elsewhere that a persistently rapid pulse uninfluenced by digitalis, indicates pericardial adhesion in the young; the same pulse uninfluenced by treatment points to coronary artery disease in the middle-aged and senile.

Myocarditis.

Acute Myocarditis. Myocarditis may be acute or chronic. The entire muscle or only a portion may be affected. General myocarditis is always acute. The local form may be acute or chronic, depending upon the degree of the primary cause. The local variety is usually due to a thrombus in the terminal endings of the coronary artery, which cuts off the blood-supply.

Ætiology. Pathological antecedents of acute general myocarditis are the infections, particularly typhoid and typhus fever, pneumonia, diphtheria, and septic infections. Chronic myocarditis is usually associated with atheroma, one of the causes of which occurs in the later stages of Bright's disease. (See Atheroma.) The result of myocarditis, when acute, is dilatation of the heart, fatty heart, or aneurism of the heart. Chronic myocarditis is followed by fatty heart, by dilatation, by the so-called fibroid heart or fibrous myocarditis, and by aneurism. The above facts in ætiology are important in diagnosis.

Symptoms. The symptoms of *acute* myocarditis are vague. In the course of, or in the convalescence from an infection the patient may complain of some oppression in the præcordia and suffer from dyspnœa; attacks of syncope may occur, and sighing may be frequent. The pulse becomes more rapid and weak, but is usually not irregular. The circulation is much depressed, the hands may be cold, the face pallid. These symptoms may be accounted for by the extreme exhaustion alone that follows fever. No doubt some myocarditis accounting for the symptoms exists in all cases, particularly if there is prolonged high temperature.

Often the patient does not complain of any cardiac symptoms. Death takes place suddenly, either in the course of the disease or after it has spent its force, from acute dilatation or cardiac paralysis. This is particularly true in pneumonia and diphtheria. In the latter affection the sudden appearance of cardiac symptoms, dyspnoea, cyanosis, and cold extremities may be due to paralysis of the heart.

Physical Signs. Enfeeblement of the heart-sounds, sometimes with accentuation of the mitral first sound, is observed. The impulse and apex-beat are scarcely perceptible or absent altogether. If acute dilatation supervenes, the area of dulness may be increased.

Chronic Myocarditis. Chronic myocarditis may exist without physical signs and may be devoid of clinical expression.

Symptoms. 1. A person may have chronic myocarditis in an advanced degree without symptoms. Death may be due to other causes, as an intercurrent affection, an infection, a terminal nephritis, or one of the many phases of endarteritis. The final termination may be "cardiac" and yet the myocarditis may not be the cause of the symptoms or of the dissolution. Thus coronary artery disease with obstruction by thrombus frequently causes death in cases of latent, and sometimes of advanced, myocarditis.

2. Often the presence of a previously unsuspected myocarditis is not made known either by signs or symptoms except in or during the course of an infection, as pneumonia, or of a toxæmia, as gout. An arrhythmia, an unsuspected dilatation, syncope, or bradycardia, may be brought out by the toxæmia or the infection.

3. The clinical expression may be effaced by the phenomena of an associated or primary valvulitis, a nephritis, an emphysema, or an arteriosclerosis.

Combined with the cardiac symptoms of myocarditis, to be referred to later, we may have the symptoms of chronic nephritis, of pulmonary emphysema, of advanced valvulitis with failure of compensation. Again, the symptoms of endarteritis in other parts may obtain, alternating in character and constancy and interchanging with symptoms of the aforementioned conditions. A cerebral thrombosis, the same condition of an extremity, uræmia, apoplexy, or perhaps phenomena of coronary artery disease (previously referred to) may close the scene. Such shifting of scenes and complexity of phenomena require a clear conception of the associated or concomitant processes in senile or sclerotic myocarditis, and intensely increase the interest in its study. It is well to dwell upon these associate and more frequently dominating features, for only by realizing the pathological lesion in the background—the endocarditis and secondary myocarditis—can the nature of the phenomena be recognized.

4. The clinical expression may, on the other hand, be simply the phenomena of senility.

5. The symptoms, singly or combined, usually the latter, compel the patient to seek relief. They are the *cardiac symptoms of myocarditis*.

1. **Heart-tire.** As seen in cardiac debility, this is out of proportion to the evidence of senile decay elsewhere. There are breathlessness, œdema of the feet, syncopal attacks or anginal attacks. Local anæmia occurs,

hence the functional activity of organs, as the brain, is in abeyance. Digestive disturbances, renal insufficiency and cerebral inactivity occur singly or combined. The syncope occurs on the slightest exertion, as straining at stool, rising up in bed, or turning from side to side; the breathlessness on any attempt to walk, often, even to use the upper extremities. Weakness out of proportion to the seemingly strong appearance of the patient is characteristic. *Tremor cordis* may be present; palpitation is not infrequent. Tachycardia is the rule and the pulse is small and feeble.

2. Dilatation of the Heart. The dropsies, the effusions, the congestions, the enlargement of organs, the functional derangements, coming and going, soon permanent, with the physical signs of cardiac dilatation, are described under dilatation. If symptoms of dilatation arise, it is unusual to have anginal symptoms. If digitalis is administered to the degree of inducing a hypertrophy, cardiac pain not unlike that of angina is caused. After long dilatation its immediate symptoms may be relieved and then those of secondary sclerosis of other organs, as of the kidneys, arise.

3. Angina Pectoris. *a.* A person with myocarditis may have one attack of angina terminating in death without premonitions of cardiac disease. They are the cases in which there is often found, associated with the myocarditis, extensive coronary artery disease, death being due to thrombosis.

b. The paroxysms may continue for twenty-four or forty-eight hours before death closes the tragic scene. Arrhythmia frequently attends the breast-pang. It may have preceded the angina for months or years. Dyspnœa might also, although not necessarily, have been a forerunner, occurring either on exertion or at rest. It is not common to see the forms of dyspnœa which will be described later, attended by shock and so often terminating fatally, associated. Nor do we usually see in these subjects of this rapidly terminating form of angina the cardiac asthma of dilatation nor the nocturnal form of dyspnœa due probably to the same cause. We do see dyspnœa from pulmonary congestion developing in the status. When the *angina vera* is replaced by *angina sine dolore*, we have asthmatic attacks in most of the cases. In rare instances the first paroxysm is followed by rapidly recurring attacks, increasing weakness of the heart, and death in cardiac asystole or the ingravescent asystole of Balfour. Houchard describes the condition as *l'état de mal angineux*.

c. The paroxysms continue for several years. During the interval the patient will suffer, as in the preceding cases, from arrhythmia and the forms of dyspnœa observed in that class. No other cardiac symptoms arise except in very rare instances. Pronounced dilatation may occur with the marked symptoms belonging thereto. On the other hand, it is usually the case that other symptoms or accidents of endarteritis do not occur. Thus an anginal subject does not die of apoplexy; again, a prolonged intermission may occur in the attacks of pain, but if arrhythmia has been present, it does not subside. The dyspnœa, except on exertion (dyspnœa of dilatation), may be in abeyance. Indeed, subsidence of the pain is usually due to moderate dilatation as indicated by the symptoms and physical signs.¹ One of my patients, suffering for one year from

¹ Musser, "Angina Pectoris," Trans. Assoc. of Amer. Phys., vol. x., p. 85.

angina, arrhythmia, and dyspnœa, and having the physical signs of myocarditis, had a severe attack of gangrenous appendicitis, for which he underwent operation, and from which he had a long convalescence, during which time he was free from pain and dyspnœa for six months, when the attacks recurred, followed by death in one of the paroxysms.

d. Occasionally we see angina *sine dolore*, at first described by Gairdner, the incident occurring in myocarditis with moderate dilatation.

4. Dyspnœa. *a.* WITH DILATATION. We see the dyspnœa of dilatation, if that condition complicate myocarditis, with or without pulmonary congestion or pleural effusion. It is constant, or it is increased by exertion, or it is paroxysmal and hence usually nocturnal. In the latter instance there are cyanosis and orthopnœa, perhaps due to a temporary increase of the pulmonary congestion. It must not be confounded with the toxic dyspnœa of nephritis so often concomitant.

It is often the first premonition of myocardial trouble; it may be during a hard pull on the bicycle or on a mountain climb. After some physical effort, dyspnœa occurs with præcordial cardiac sensations of fullness or of sinking or with palpitation or syncopal sensations. After a brief rest, it may disappear and not recur for months, or a slight exertion again brings it about. Frequent recurrences become a warning, and very soon arrhythmia, angina, or the congestions of dilatation ensue. At first it may be due to temporary acute dilatation or to arrhythmical action of the papillary muscles with asystole.

b. MYOCARDIAL DYSPNŒA. Dyspnœa associated with the aforementioned localized substernal distress without true paroxysms of angina. Often, in your presence, the patient will have paroxysms, probably induced by the psychical excitement attendant upon the consultation. A paroxysm occurs independently of such influence, however, possibly following the taking of food. The patient takes ineffectually breath after breath without satisfying air-hunger until a final full breath satisfies him for a time. "There," remarked M., after repeated labored breathing, "I have gotten over it. I can now breathe easily." The patient's features are such, with the ashy countenance and the cardiac signs, as point unmistakably to the myocardial condition. One can almost make a diagnosis by the facies and the intermittent dyspnœa. In many of these cases the dyspnœa is relieved after some physical exertion. It must not be confounded with the curious form of psychical dyspnœa occurring in certain forms of indigestion, from gastric neurasthenia.

c. DYSPNŒA WITH ANGINA. The often-repeated story, "I walk half a square and then must seek support to get my breath and relief to the breast anguish." Usually if the angina occurred when at rest there was no dyspnœa. This was not always the case, however, and the interruption to breathing was created by, or coincident with anginal paroxysms. This is one of the true forms of myocardial dyspnœa.

d. DYSPNŒA DUE TO ASYSTOLE. Angina is one of the tragic terminations of a career which has been fraught with the high and often severe duties in the world's battle. Another tragic end is that attended by the paroxysms of dyspnœa and shock we are about to describe. Often called "heart-failure" or "œdema of the lungs" or, wrongly, "conges-

tion of the lungs," one paroxysm frequently ending the battle. Again, the attacks may recur at intervals of weeks or months, the fatal paroxysm not occurring until a year or two after the original attack. I have seen this continue in rare instances over three years. In one patient, a woman aged seventy-nine, the attacks recurred for five years.

The attacks are characteristic. The patient is suddenly seized with dyspnœa. The respirations are labored, but not much increased. A rattling sound attends it, readily found to be due to fluid in the tubes, as the general large and small râles show. The clear fluid pours from the mouth or is discharged easily, if the attack is not to be fatal; with difficulty or not at all, if death is imminent. The respiratory distress is extreme. The patient sits upright; the face is pale; the brow is covered with sweat; the hands are cold and pallid. There is little if any lividity. The temperature is subnormal; the pulse rapid, thready, irregular, perhaps not perceptible at the wrist. The patient is in a state of profound shock. Because of the lung-noises, the heart-sounds can not be heard, or only as a confused jumble, or with irregular and ineffectual systoles. Either no change in the cardiac dulness is distinguished or it may be lessened.

In the interval, after full recovery, the patient is fairly free from dyspnœa. There is probably some arrhythmia; rarely any angina, and dilatation does not supervene. As in angina, the occurrence of one attack is usually the beginning of the end. I have never seen any patient thus affected live more than five years, and usually death occurs within the year from the onset, generally from an attack similar to the original, such attacks having grown more frequent toward the end. Of course, a progressive weakness develops, and nephritis may become a dominant feature.

These attacks of cardiac asystole differ from those of acute dilatation. The difference is mainly in the following particulars. Had the patient been examined before the attack, signs of myocarditis would have been present in the acute dyspnœa of that affection, whereas dilatation could occur without the presence of fibromyocarditis. In the former, shock and œdema of the lungs are dominating features. In dilatation, marked cyanosis is prominent, tachycardia and arrhythmia are extreme, while the lungs are the seat of congestion rather than œdema. In dilatation there is congestion of the spleen, liver, and kidneys. The urine gives evidence at once of the congestion, whereas it is not necessarily nor instantly changed in myocardial dyspnœa.

It seems academic to discuss the diagnosis of œdema and congestion of the lungs, for it is usually not practicable. It must be admitted that very frequently it can not be done. Nevertheless, it seems well to attempt it, for, as one appears to be a feature of myocardial dyspnœa (œdema) and the other of dilatation (congestion) and as the treatment of each is different, it is well to have an idea of their distinction. I may say in passing that, as I have stood at the bedside of these cases, my feelings were that the œdema was largely from impairment of innervation of the pneumogastric nerve, whereas the pulmonary congestion was vascular.

œdema of the lungs is attended by shock, by an abundant, frothy expectoration, by dyspnœa without much cyanosis, and by signs of the

outpouring of fluid in the large bronchi rather than in the air-vesicles. Congestion of the lungs is attended by cyanosis, by some cardiac distress, as palpitation, by congestion of other organs, and by the physical signs of moderate filling of air-vesicles with fluid and by frothy, bloody, but not abundant expectoration. There does not seem to be the same fear of impending death in dilatation. While air-hunger is the same in both—orthopnœa marked—it is a necessary attendant of myocardial dyspnœa, but not of the congestion of dilatation.

5. Tachycardia. Frequently an unduly rapidly acting heart is due to myocarditis. Usually there is some arrhythmia, but it is remarkable how little suffering may occur, although the heart is beating at the rate of 150 to 180 per minute. Such heart-hurry must be distinguished from toxic and from reflex forms of tachycardia. It must be remembered that it is not to be confounded with palpitation, the phenomena of which give rise to actual mental suffering and to local distress. It is one of the essential symptoms of Graves' disease and a common accompaniment of mitral stenosis. Even in the absence of these conditions tachycardia may be a symptom of myocardial strain, and may be relieved by judicious management. The great fact to be remembered is that tachycardia may be the terminal cardiac symptom of myocarditis, death resulting in from forty-eight hours to one month after the onset, the end being attended by *ingravescent asystole*, congestions of the internal organs proceeding slowly to embarrass further the causal failing circulation. A patient with tachycardia may suffer from *tremor cordis*, the sensation of fluttering followed by a systolic shock belonging thereto alone giving rise to distressing symptoms.

6. Bradycardia. Time forbids a description of the interesting syndrome of Stokes-Adams. It is now well recognized that with this slow pulse, syncope, convulsions, and pseudo-apoplexy may arise. Patients with this combination of symptoms rarely suffer from angina or from severe arrhythmia. A fatal syncope is the cause of death. A progressive dilatation rarely takes place. The syncope may be hastened by exertion or by excitement.

7. Arrhythmia. Finally, myocarditis may have clinical cardiac expression in arrhythmia alone. Palpitation may attend, but does not commonly do so; delirium cordis, however, is not infrequent, and death may come after many years from asystole, sudden or ingravescent. In the marked cases early in the ailment it is not uncommon to see dilatation with dropsies, congestion and the physical signs of mitral insufficiency. Improvement may take place for a time, followed by recurrences, and death occur from either nephritis, dropsies and cardiac asthenia, or from a terminal infection. Death from angina is rare; indeed, occurrence of the paroxysms is rare. Dyspnœa, however, is common when signs of dilatation arise.

Objective Diagnosis. The most common objective sign is the sallow, pallid complexion. This is most striking. When such persons present themselves, it is safe to infer that the endarteritis has involved the coronary arteries and that the myocardium has suffered.

With the sallow countenance premature age manifests itself in the color of the hair, the baggy eyelids, and the abundance of wrinkles.

Physical Diagnosis. *The Arteries.* The palpable, often tortuous vessels show thick walls and high tension. The temporal arteries are prominent and the other indication of degeneration, *arcus senilis*, is present. The pulse is like that of endarteritis. The artery is readily palpable, isolated with ease, showing high tension, and while apparently strong because of a quality of fulness which with the hard vessel gives a feeling of strength, yet it is actually feeble. The arteries are fibrous but not necessarily atheromatous. Indeed, the patient with pronounced atheroma will probably not have symptoms at least of endarteritis and will die of cerebral thrombosis or of senility alone, or of some intercurrent or terminal infection. Our middle-aged patient with endarteritis does not necessarily end with advanced atheroma, but dies of apoplexy, nephritis, angina, or sudden heart asystole.

The Heart. The physical signs of the heart are those of the (a) myocarditis alone, or those of (b) myocarditis plus some hypertrophy, or those of (c) myocarditis plus dilatation.

The physical signs of *myocarditis* are those of feeble or absent impulse; or if palpable, of apex-impulse displaced to the left; of marked increase in the area of absolute cardiac dulness and of characteristic auscultatory phenomena. The latter phenomena are those either of a systolic shock, greater than the force of the impulse would lead us to believe to be present, or of feeble muscular sound. In the early stages the former exists, and continues later if hypertrophy coexists. From the first, or at least early, we have gallop rhythm, or reduplication of the systolic sounds. This reduplication may be heard over the right heart or more distinctly over the left heart; sometimes it is heard all over the præcordia. My impression is that it is more commonly heard in the parasternal line at about the fourth rib. It may be more marked in the supine position and is generally more marked after exertion. It may disappear after a stimulant is taken or if the heart is stimulated by fever. Reduplication of the second sound also obtains, but is less frequent in the myocarditis of coronary artery disease than in that due to valvulitis or nephritis. In uncomplicated cases murmurs are not heard until late in the disease. Sometimes, however, we hear a systolic murmur at the fourth rib, greater in the recumbent posture. This murmur is soft, low in pitch and so often heard in the parasternal line that it is my custom to call this line the myocardial line. However, it may be at the tricuspid or even in the pulmonary area—when it is probably, although not necessarily, hæmic.

The papillary muscles are the source of the auscultatory phenomena of myocarditis whereby leaflets do not coapt synchronously, or areas of the muscle contract asynchronously.

When *dilatation* supervenes, the physical signs change in keeping with the physical condition of the heart. The auscultatory signs of insufficiency at the mitral and tricuspid orifices are predominant, rarely at the aortic; accentuation of the pulmonary second sound is a frequent sign, with low-tension pulse and engorgement of the venous side of the circulation.

The myocarditic heart of *fatty degeneration*—the fatty heart—presents some similar signs. A visible impulse is wanting and any palpable im-

pulse is felt as a slap against the chest-wall. The area of dulness is normal or slightly increased; a loud hæmic murmur at any orifice, but most frequently at the pulmonary and aortic valve, may be heard. Just as frequently the sounds are almost inaudible, only the second sound at the base being conducted to the ear. There is pulsation of the vessels of the neck out of proportion to the apparent force of the heart.

We may say, then, that myocarditis may exist (1) without definite physical signs—a rare occurrence; (2) with signs of moderate cardiac hypertrophy, marked reduplication being the only physical sign and of significance only when coupled with signs of endarteritis; (3) with physical signs of dilatation; (4) with physical signs of fatty degeneration; (5) with the physical signs mentioned, which in the aggregate are of great significance.

Aneurism of the Heart.

Aneurism of the valves, following endocarditis, can not be recognized during life. Aneurism of the walls usually results from chronic myocarditis. The aneurism develops in the left ventricle at the apex. The symptoms are indefinite. In rare cases a marked bulging has been noted in the region of the apex, and the tumor may perforate the chest-wall. A projection beyond the normal line of cardiac dulness may be detected by stethoscopic or plessimetric percussion. The symptoms are those of myocarditis and of dilatation of the heart. (See Diagnosis.)

Rupture of the heart is one of the causes of sudden death, often without previous symptoms. The accident takes place during exertion. Quain collected 100 cases, in 71 of which death took place without warning. In other instances there was a sense of anguish and suffocation in the cardiac region. The physical signs of slowly developing pericardial effusion may be obtained if the leakage from rupture is slow in progress.

Chronic Valvular Disease.

Valvular disease includes valvulitis and valvular incompetence; there is either obstruction or regurgitation at the orifices affected. Valvulitis may exist with or without symptoms; valvular incompetency is always accompanied by symptoms. Valvulitis implies organic disease of the valves; valvular incompetency indicates regurgitation through orifices whose valves, whether healthy or diseased, are unable to effect perfect closure.

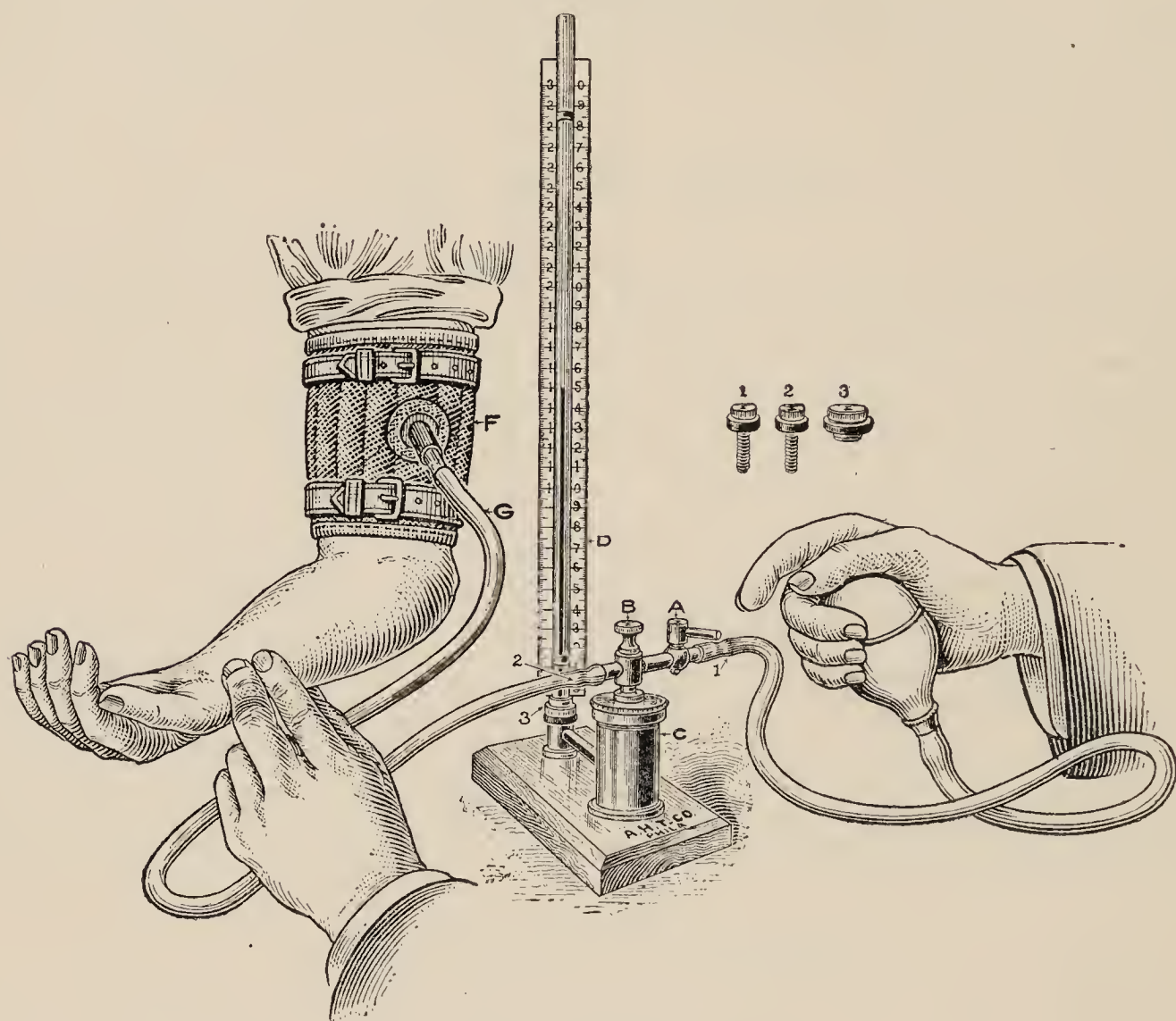
Valvulitis may be recognized by physical signs of (1) the lesion, (2) the secondary effects of the lesion on the heart and circulation—hypertrophy or dilatation.

Valvular incompetence occurs usually in dilatation, and may be secondary to valvulitis. It is recognized by both signs and symptoms. Valvular disease is without symptoms so long as the heart muscle enlarges sufficiently to keep in balance the impaired circulation; compensation is then said to be complete. When compensation is broken, we have the subjective symptoms enumerated above, all in consequence of dilatation

of the heart. It may be said that valvulitis is of no significance so long as compensation is perfect.

To review—valvulitis may be attended by physical signs in the heart and vessels only, or by its own physical signs, the physical signs of dilatation, and the symptoms of the latter. In the consideration of valvular disease it is more profitable to take up the symptoms of each valve-lesion, bearing in mind that two or more of the valves may be diseased at the same time, or that both obstruction and regurgitation may be present at the same time at the same valve-orifice. The severity of the valvular

FIG. 349.



Stanton's blood-pressure indicator.

defect, as well as the amount of work performed by the heart and coincident strain on the arterial system may be estimated accurately by means of an instrument that has appeared recently and which is shown in the accompanying illustration (Fig. 349).¹

¹ With this apparatus, which is constructed on the general plan of the Riva-Rocci instrument (see Fig. 106 and p. 415), the highest (systolic) pressure is obtained by noting the height of the mercurial column at the moment when the pulse reappears after compression, while the lowest (diastolic) pressure corresponds to the point on the scale at which the greatest oscillations occur in the mercurial column. This instrument differs from others of its kind chiefly in the greater width of the compressing armlet, which is much broader than any hitherto employed; the mode of attachment of the manometric tube secures a firm and unyielding support; the rubber tubing is non-distensible; and the manometer is of special construction. The rubber armlet H ($3\frac{1}{4}$ inches wide) is prevented from expanding outward by a cuff, F, of double-thick canvas, reinforced by the insertion of tin strips, and is held by two straps which completely encircle the compressing band. The arm-piece is applied above the flexed elbow by overlapping the ends smoothly. The rubber armlet is connected by stiff-walled tubing, G, to the manometer. The manometer

Aortic Regurgitation: Insufficiency or Incompetence. This may exist for a long time without presenting any symptoms. It occurs more frequently in men than in women, and is more common in the later periods of life. It may be due to congenital malformation, to acute endocarditis, or, as is most frequently the case, to chronic endocarditis, particularly when it follows strain or undue exertion. Alcoholism and syphilis are also frequent antecedents. In rare cases it follows rupture of the valves. Relative insufficiency or incompetence is of very rare occurrence. Insufficiency is frequently combined with obstruction.

The blood falls directly into the left ventricle during the diastole. There is, first, a relative diminution in the amount of blood in the artery; and, second, an increased amount of blood in the ventricle, because the regurgitated column of blood meets the blood from the auricle which is filling the chamber at the same time. Dilatation of the left ventricle ensues, and is followed by hypertrophy. Dilated hypertrophy thus arises. The heart becomes enormously enlarged. This is one of the conditions in which enormous cardiac enlargement takes place—so-called *cor bovinum*. If this valve-lesion occurs late in life and from the above mentioned causes, it is attended by more or less sclerosis of the arteries.

Symptoms. They may be entirely absent so long as perfect compensation exists. This is particularly the case when there is but little general arterial sclerosis. Coincident lesions of other valves tend to break the compensation. The earlier symptoms are those due to arterial anæmia, particularly anæmia of the brain. They are headache, dizziness, and flashes of light before the eyes. The patient has an anæmic appearance, and soon begins to suffer from shortness of breath. This at first develops upon slight exertion. Palpitation and oppression about the chest are complained of, readily excited by undue exertion. Pain is a common symptom. It may be in the region of the præcordia, of a dull, aching character, and radiate to the neck and down the arms, particularly on the left side. The anginoid pains may be followed by attacks of true angina pectoris. The latter are more common in aortic regurgitation than in any other valve-lesion.

consists of the metal chamber C, and a horizontal metal tube inserted into its base, to which is screwed the vertical glass tube D, the calibre of which is approximately $\frac{1}{100}$ the calibre of C. The cap of C, which screws on, is provided with a metal T, one end connecting with the armlet and the other with the rubber-bulb, while the vertical limb opens into C. The stopcock A shuts off the rubber hand-bulb from the rest of the system, the screw-valve B allowing gradual escape of the air. By removing the vertical glass tube and screwing in the caps 1, 2, and 3, as indicated in the figure, the instrument is rendered transportable.

The armlet and cuff having been applied, the valve B is screwed tight and the cock A opened (arm parallel to limb of T). With the fingers of the left hand on the pulse, the rubber bulb is held in the right hand in the manner shown, leaving the thumb and index finger free. The bulb is repeatedly compressed until the pulse is no longer palpable, when, with the thumb and index finger of the right hand, the cock A is closed (turned at right angles). The screw-valve B is now slowly turned, and with the escape of air the mercurial column falls. The point at which the pulse reappears is mentally noted as the *high* or *systolic pressure*. At or even above this point oscillations appear in the mercurial column which increase as the column falls until they reach a maximum, and then again diminish. The pressure at which the greatest oscillation occurs is noted as the diastolic pressure. By unscrewing the valve B completely all the air is allowed to escape from the apparatus.

(See also "A Practical Clinical Method for Determining Blood-pressure," etc. William B. Stanton, University of Pa. Med. Bulletin, Feb., 1903, and the discussion.)

As *compensation* fails, venous stasis occurs and the dyspnœa increases. The latter is worse at night and compels the patient to sleep in a semi-erect posture. Congestion of the lungs takes place, giving rise to cough. Hemorrhage occurs, but not so frequently as in mitral disease. Œdema of the feet sets in, but general anasarca is not common. Œdema of the feet may be due to the attendant anæmia.

In aortic insufficiency *sudden death* is of common occurrence. This may take place at night during an attack of dyspnœa, or occur suddenly upon the slightest exertion, such as straining at stool, or ascending a height, or walking more quickly than usual.

Physical Signs. (Plate XXXI., Fig. 1.) **INSPECTION.** The apex-beat is downward, outward, and to the left. It may be as low as the seventh interspace, and as far out as the anterior axillary line. The area of cardiac impulse is increased. It occupies the whole præcordia, and heaving of the lower half of the chest may be seen. In young subjects there is præcordial bulging.

PALPATION. The impulse is strong and heaving. After compensation fails it is indefinite and wavy. A thrill, diastolic in time, may be felt if the hand is placed above the middle of the sternum.

PERCUSSION. The area of dulness is increased. The extent is greater than that in any other valve-lesion, and the enlargement is more particularly downward and to the left.

AUSCULTATION. At the second costal cartilage on the right a murmur is heard, *diastolic* in time. This may be its seat of maximum intensity. (See Fig. 128.) It is transmitted along the course of the sternum toward the apex. In some instances the seat of maximum intensity is at the fourth left costal cartilage, or even at the apex. The second aortic sound is absent in the large majority of cases. However, both murmur and second sound may be heard at the same time.

ASSOCIATE MURMURS. Other murmurs also may be associated with aortic regurgitation, not always due to disease of the aortic valves :

1. A *systolic* murmur at the second costal cartilage on the right, transmitted into the vessels of the neck, short, rough, and high in pitch. It is due to roughening of the valve-segments, to atheroma of the aorta, to aneurismal dilatation of the aorta or associated aortic stenosis.

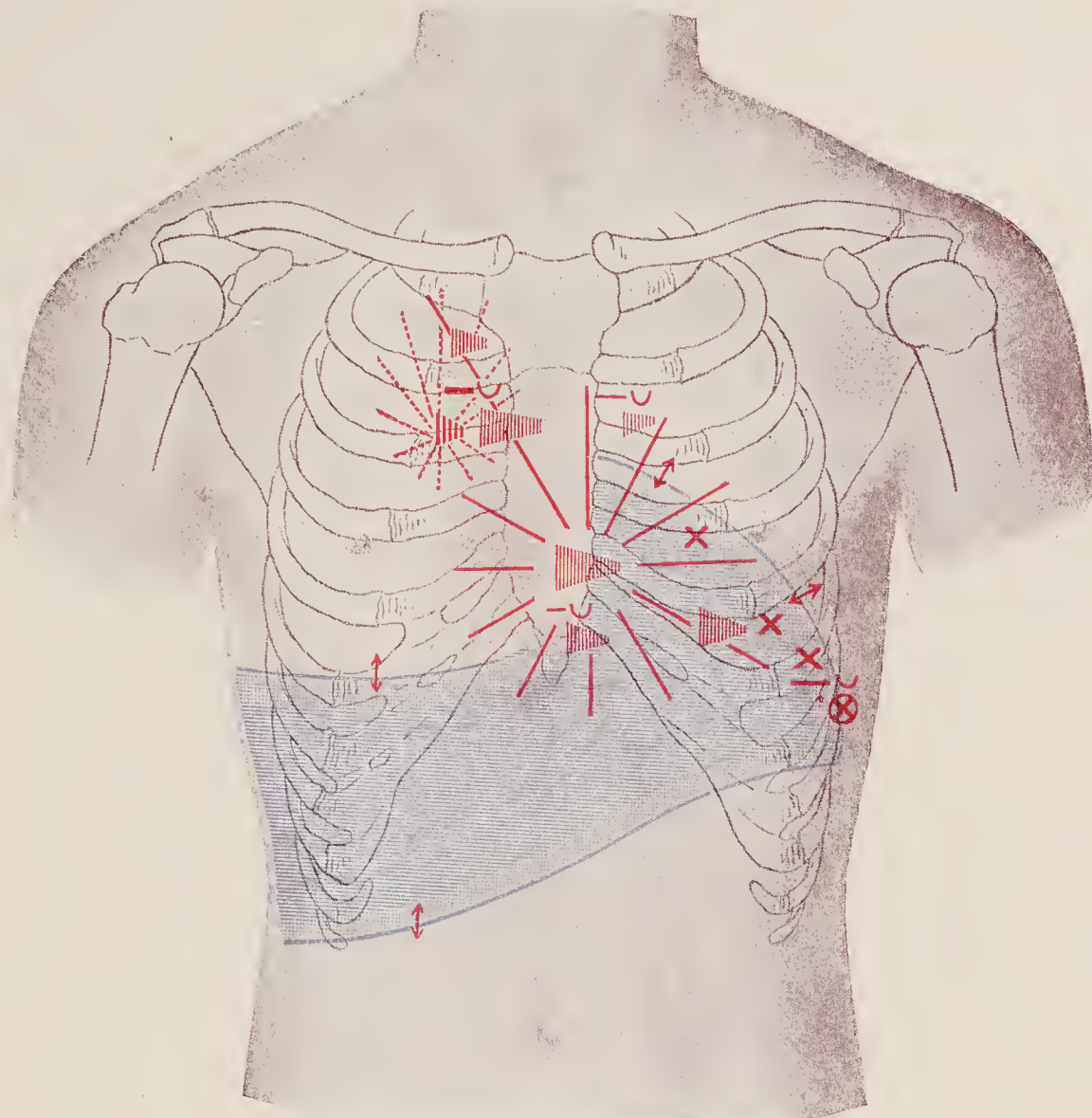
2. *Flint's Murmur.* A murmur at the apex, rumbling in character, localized to this area, usually *presystolic* in time. It is the murmur described by Flint, who attributed it to flapping of the mitral segments, which during diastole are not forced back against the heart-wall, on account of the dilatation of the ventricle. They remain in the blood-current and produce relative narrowing.

3. A *systolic* murmur in the mitral area, low in pitch, due to dilatation of the ventricle and consecutive incompetency of the mitral valves. This occurs when failure in compensation takes place.

EXAMINATION OF THE ARTERIES. Pulsation of the peripheral vessels is more common in aortic regurgitation than in any other valve-lesion. The carotids throb, the temporals pulsate, the brachial and radial arteries are conspicuous. Pulsation of the retinal arteries is seen with the ophthalmoscope, and has often led to recognition of the disease

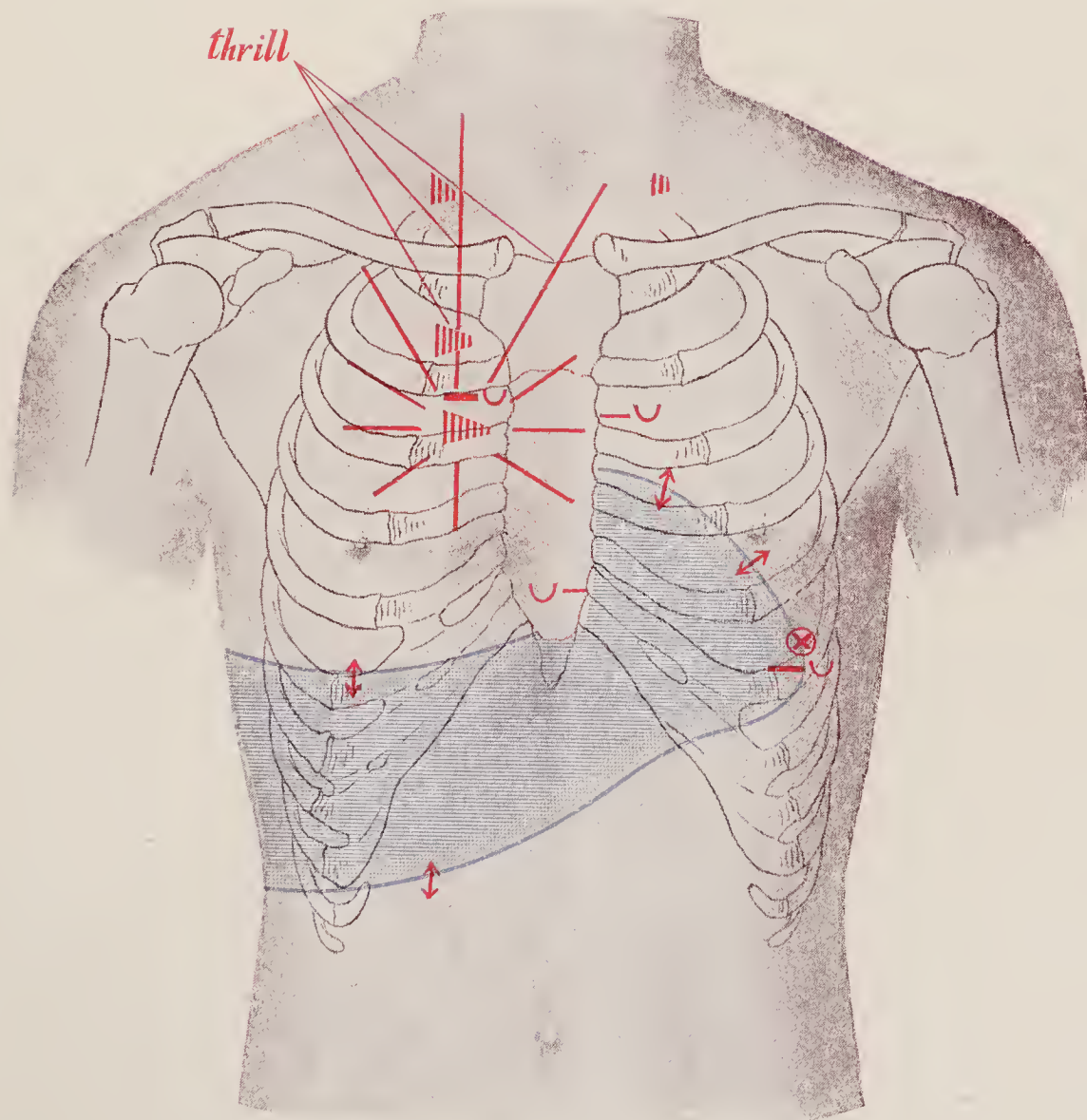
PLATE XXXI.

FIG. 1.



Aortic Regurgitation.

FIG. 2.



Aortic Obstruction.

by the ophthalmologist who had been consulted for other conditions. Pulsation of vessels in the fauces is occasionally seen. The pulsation is of a jerking character; in the neck it may simulate the pulsation of an aneurism. The aorta can be seen and felt at the suprasternal notch. The abdominal aorta pulsates vigorously in the epigastrium.

The *pulse* is significant in aortic regurgitation. The so-called water-hammer, or Corrigan's, pulse is observed. The pulse is quick, of large volume, and jerking, and after striking the finger immediately recedes. It is most marked when the arm is extended vertically.

On *auscultation* of the arteries double murmurs may be heard in the carotids and subclavians, and in rare instances they are present in the femorals. (See Pulse.)

THE CAPILLARY PULSE. This is seen beneath the finger-nails, or in the mucous membrane of the lips, or on the surface of the skin, as the forehead, when a line is drawn across it. The hyperæmia produced on either side of the line becomes alternately red and pale. Capillary pulse also occurs in anæmia, and at times in neurasthenia.

Diagnosis. No valvular lesion is more easily recognized when the arterial symptoms and signs are also present. The interpretation of a diastolic murmur at the base of the heart is difficult. It may be due to congenital disease, to aneurism, or to conditions of relaxation, as in anæmia or when there is unusual loss of tone in the bloodvessels. Cabot has noted a diastolic murmur, as seen in aortic regurgitation, in cases of anæmia, in adherent pericardium, and in dilatation of the aorta; and I have seen it come and go in exophthalmic goitre and in myocarditis.

Aortic Obstruction. Aortic obstruction occurs in the aged, and with atheroma of the arteries. It causes some diminution in the amount of blood in the peripheral circulation, resulting in poor nutrition and the development of anæmia.

Symptoms. Anæmia develops first, and embolic phenomena may occur later. When the obstruction is pronounced, the blood-supply in the arteries is diminished. Cerebral anæmia takes place, causing dizziness and fainting. Sleep is more disturbed than in other valve affections, because of the cerebral anæmia.

EMBOLIC SYMPTOMS. The symptoms may be latent until the occurrence of embolism. This accident is not uncommon, on account of the position of the aortic valve. The emboli are distributed throughout the arterial circuit, and may lodge in the brain, kidneys, or spleen.

CARDIAC SYMPTOMS. Palpitation and cardiac pain occur, but are not so common as in aortic regurgitation. When compensation fails, dilatation of the left ventricle ensues, followed by pulmonary congestion and stasis in the systemic circulation.

Physical Signs. (Plate XXXI., Fig. 2.) There is hypertrophy of the left ventricle.

INSPECTION. The apex-beat is displaced downward and outward. The impulse is strong during the period of hypertrophy. When compensation fails, the physical signs of dilatation ensue. In many cases, from the very first there may be considerable hypertrophy without the visible impulse,

because of associated emphysema, which is common in old men with this lesion.

PALPATION. At the base of the heart and in the aortic area a thrill, systolic in time, may be felt. When present, it is usually very distinct, and is transmitted along the course of the vessels. The impulse is slow and heaving, if hypertrophy is present; if dilatation, feeble and indistinct.

PERCUSSION. In the earlier stages the area of dulness is increased to the left and downward. After compensation is broken, dilatation with increased area of dulness ensues.

AUSCULTATION. A murmur is heard of maximum intensity at the second costal cartilage to the right, *systolic* in time, and transmitted in the course of the bloodvessels. (See Fig. 125.) It is usually harsh and loud, but may be musical. As the heart weakens, the intensity of the murmur lessens and its roughness disappears. It becomes soft and low in pitch. The second sound, if there is no regurgitation, is muffled or may be absent. The *pulse* is slow, small, and regular (*pulsus tardus*). The tension is usually increased.

Diagnosis. A systolic murmur at the aortic orifice may be due to aortic obstruction, atheroma or dilatation of the aorta, ulcerative aortitis, or anæmia. Such a murmur is also common in aortic insufficiency without narrowing of the orifice, and is due to stiffened valve leaflets projecting into the blood-current. Huchard describes a murmur in this situation, with vibratory thrill, due to aberrant chordæ tendineæ. The murmur of aortic stenosis is distinguished from the others by its character, by the presence of thrill, by the character of the pulse, and by its association with hypertrophy of the left ventricle. A murmur due to atheroma of the aorta, particularly in the course of renal disease, is also associated with hypertrophy of the left ventricle. The diagnosis from aortic obstruction is often difficult or impossible. Slowness of the pulse is more characteristic of aortic obstruction. The murmur of anæmia is softer and low in pitch. There is no thrill, and the left ventricle is not hypertrophied. Anæmic murmurs may be heard elsewhere. In atheroma the second sound is usually accentuated, and in anæmia also it is intensified.

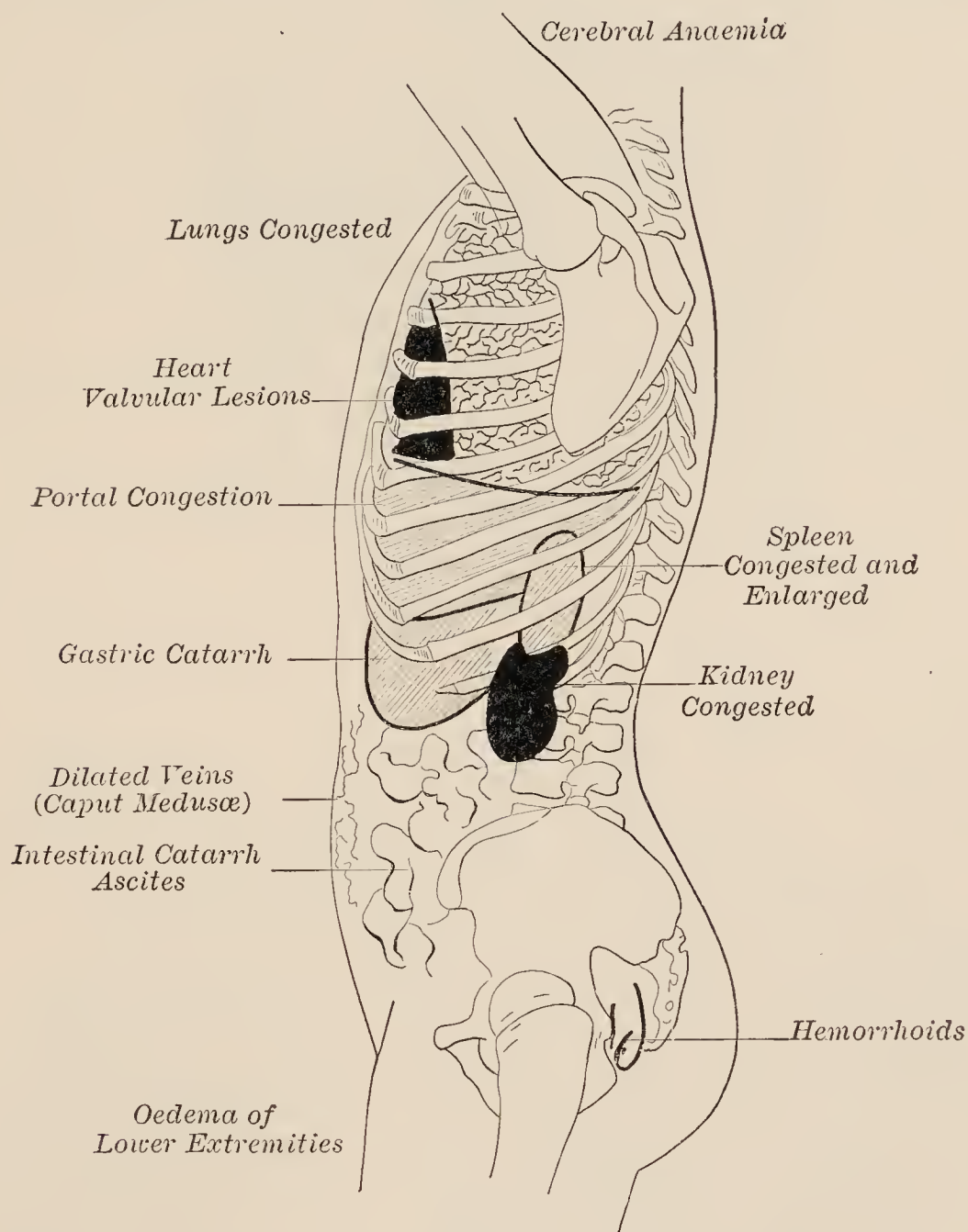
Mitral Regurgitation. The regurgitation may be due to disease of the valves (organic) from previous endocarditis; or to inability of the segments to close the orifice (incompetence), enlarged from dilatation. Incompetence occurs in dilatation of the left ventricle. It takes place when the muscle is weak in fevers and in anæmia. It is thus seen that the murmur of mitral insufficiency is one of the most commonly observed of all valve-murmurs. Its ready production and often equally ready removal with treatment make it the least serious. It must not be forgotten that insufficiency from disease of the valves and from disease of the muscles must, if possible, be distinguished from each other. The history of the case is essential in determining the diagnosis.

Mitral insufficiency or regurgitation has more serious effect upon the pulmonic and arterial circulation than any other valvular disease. These effects must be understood in order to appreciate the symptoms of mitral incompetence, which are as follows:

1. With each systolic contraction, on account of the insufficiency of the mitral leaflets, the blood flows back to the auricle, where it soon meets a volume of blood coming from the lungs. The combined volumes of blood over-distend the auricle. Dilatation ensues, and because of increased work to get rid of the increased contents, hypertrophy follows. Dilated hypertrophy of the left auricle is the first effect.

2. As a result of the above, a larger amount of blood is forced from the left auricle into the left ventricle; in order to remove the increased amount of fluid, dilatation and subsequent hypertrophy of this chamber follow.

FIG. 350.



Showing the congestions following valvulitis with failure of compensation.

3. On account of the over-distended auricle the pulmonary veins are not fully emptied during the diastole of that chamber. The veins are therefore engorged and interfere with the flow of blood through the pulmonary circuit. In consequence of the impeded flow of blood the vessels in the pulmonary circuit are dilated and over-distended. The right ventricle is compelled to act more vigorously, and even then can not empty itself freely. Dilatation and hypertrophy of the right ventricle ensue.

4. This condition causes obstruction to the flow of the blood from

the right auricle to the right ventricle : dilatation and hypertrophy of the right auricle follow.

If perfect *compensation* ensues through hypertrophy of both ventricles, engorgement in the lungs may not be observed. Moreover, the left ventricle is allowed to send out sufficient blood to supply the wants of the system. This compensation may continue for years. If it fails, either from increase in the valve-lesion, or valvular incompetence, or from weakening of the muscle, a normal amount of blood is not distributed throughout the aortic area, but is thrown back upon (1) the left auricle ; (2) the pulmonary circulation ; (3) the right heart ; and (4) the systemic veins. For a time the pulmonary circuit alone is engorged, subsequently the systemic veins become congested because of dilatation of the right auricle and incompetence of the tricuspid valves. We then have the secondary effects of stasis upon the various organs of the body, with cyanotic induration and the development of dropsies.

Mitral incompetence without disease of the valves is of frequent occurrence in emphysema of the lungs and in Bright's disease, and is a condition which always attends hypertrophy and dilatation, or may take place from various causes. (See Hypertrophy and Dilatation.)

Symptoms. As to the general symptoms, in a large number of cases perfect compensation may continue for a long time ; no subjective symptoms arise, nor are there symptoms due to dilatation.

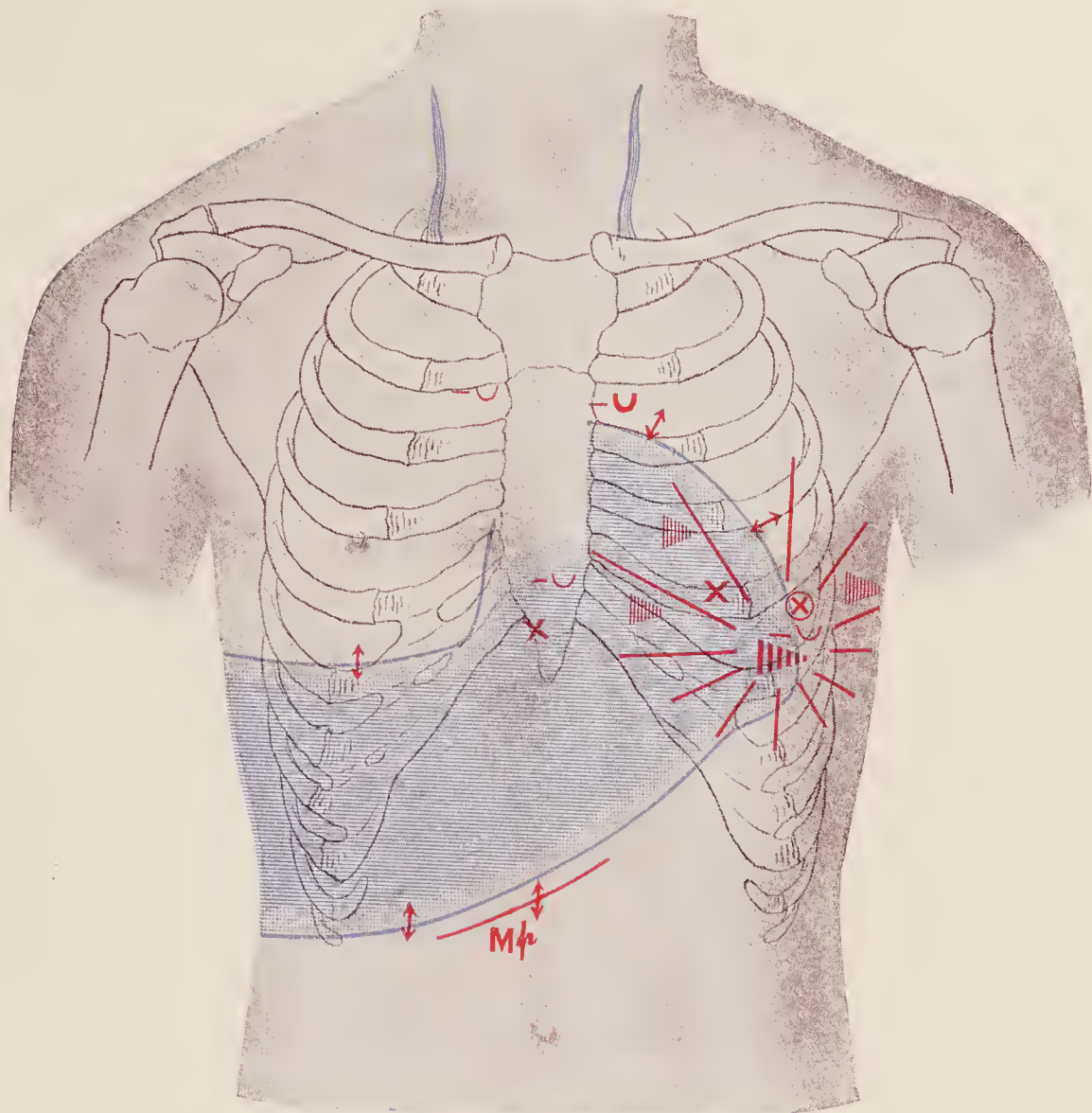
Compensation Moderate. If compensation is not perfectly effected from the first, or is broken suddenly or gradually, the symptoms of dilatation arise. In patients in whom compensation remains only fairly good we have the characteristic appearances of heart disease. It is to this class of patients that the general descriptions of heart disease apply. The face is pale and pinched, the lips and ears dusky, the capillaries of the cheeks enlarged, the finger-nails clubbed, particularly in children ; shortness of breath on exertion may be the only symptom complained of, and this may exist for years. The patients are, however, liable to attacks of bronchitis and of pulmonary hemorrhage.

Cardiac Symptoms. Compensation Lost. Palpitation may occur in this as in other forms of heart disease, and from the same cause. When the compensation is broken, symptoms referable to the heart and to engorgement of systemic and pulmonary veins occur. Of the former, palpitation with a sense of oppression is the most common ; pain is rare.

Venous engorgement leads to *congestion, cyanosis, and dropsies*. We now have the symptoms of dilated right heart superadded. The lungs are the first to be congested. Dyspnœa becomes constant and is aggravated by exertion. Cough is present, excited by exertion or speaking. With the cough there is bloody expectoration. Cyanosis occurs, and congestion of other organs follows. The liver is enlarged ; obstruction in the portal area is prominent ; chronic gastritis or gastro-intestinal catarrh ensues. The spleen is enlarged ; ascites develops, and hemorrhoids and congestion in the rest of the portal area are seen. The kidneys are congested ; the urine is scanty, albuminous, and contains casts and blood-corpuscles. At the same time that the internal viscera are congested dropsies take place, beginning in the feet and extending to

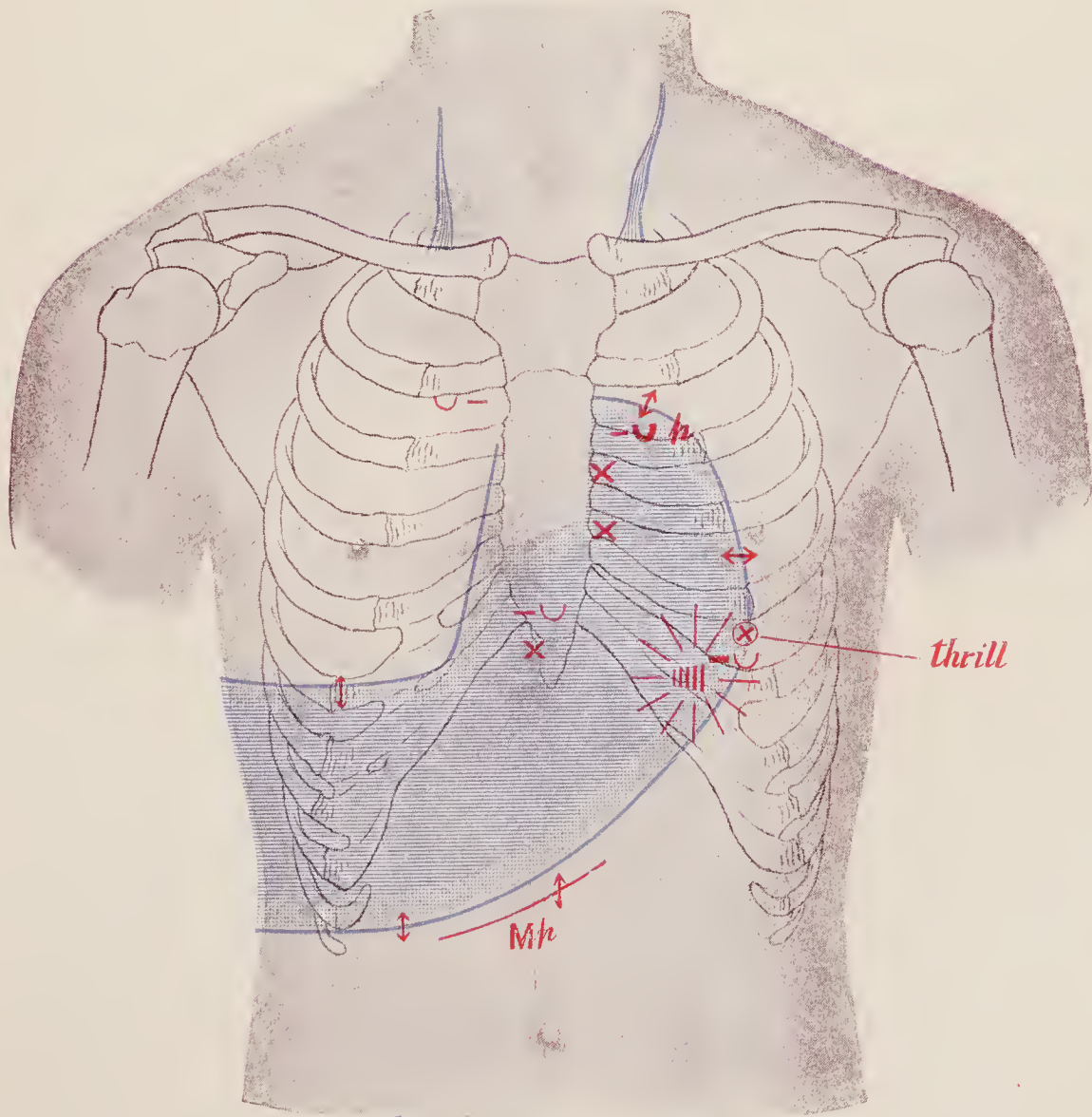
PLATE XXXII.

FIG. 1.



Mitral Regurgitation.

FIG. 2.



Mitral Stenosis.

the rest of the body. Dropsy may have been present in the feet before symptoms of portal congestion ensued.

The patient may be relieved and compensation continue for a long time. Frequent attacks of dilatation of this character may take place, their recurrence being due to lack of care in hygienic matters, or failure of health from other causes. Finally, however, the compensation can not be restored; the stases persist; the dropsies become more marked, and the symptoms of cyanotic induration and secondary scleroses of the internal organs follow. It must not be forgotten that this is the chief form of organic heart disease seen in children.

Physical Signs. (Plate XXXII., Fig. 1.) **INSPECTION.** On inspection the præcordial area appears prominent; the apex-beat is displaced to the left and rarely downward. It may extend beyond the anterior axillary line. The cervical veins pulsate and are distended. The area of impulse is increased.

PALPATION. The character of the impulse depends upon the stage of the disease at which the case is examined. At the time of full compensation it is strong and even; when this is broken, it is feeble and diffuse. A thrill is uncommon; but when present, is systolic in time and felt at the apex.

THE BLOODVESSELS. The amount of blood in the arteries is diminished. There is notable absence of visible pulsation in the arteries. The pulse at first is full and regular. It is notably small in volume and soft. As soon as failure of compensation takes place the pulse becomes irregular. The irregularity may be that of time as well as of volume.

PERCUSSION. The area of dulness is increased transversely, but especially to the right. The transverse diameter of the heart is much increased because of dilatation of both chambers. The area of dulness may extend beyond the right margin of the sternum to the extent of an inch or more and to the left as far as the midclavicular line, sometimes to the anterior axillary line. The cardiohepatic triangle is preserved.

AUSCULTATION. At the apex, the mitral area, a murmur is heard. The point of maximum intensity is in this region. It is *systolic* in time; and may replace the first sound entirely. It may be soft and low in pitch, or rough, high in pitch, even musical in character. It is transmitted to the axilla and the angle of the scapula. (See Fig. 123.) In some instances it may be heard loudest along the left border of the sternum. The pulmonary second sound is accentuated; the accentuation is loudest in the pulmonary area at the second left interspace. It may be very loud over the right ventricle, between the parasternal line and the left edge of the sternum. The murmur of mitral insufficiency is modified by the position of the patient and intensified after exertion. It may be present when the patient is lying down, and disappear in an erect posture. It may disappear when the patient is quiet and return after exertion. Other murmurs are sometimes heard:

1. A presystolic murmur, soft or rumbling, due to associated mitral stenosis.
2. When dilatation ensues, a low-pitched systolic murmur is heard at the ensiform cartilage and at the lower left border of the sternum. It is due to tricuspid regurgitation.

Diagnosis. Of special diagnostic significance are : the position of the murmur and the direction of its transmission ; accentuation of the pulmonary second sound ; enlargement of the transverse diameter of the heart, due to dilatation of both ventricles.

The diagnosis is usually easy if the physical signs are sought for. Very often examination of the heart is neglected, and the patient is treated for the symptoms that arise from congestion of the viscera. We have often seen chronic gastritis or gastro-intestinal catarrh, due to mitral insufficiency, not relieved because the primary lesions had not been ascertained. In the same way cardiac cough or dyspnoea may be overlooked. It is important in the diagnosis to determine, if possible, the nature of the insufficiency, whether it is due to disease or incompetency of the valves. As previously mentioned, the history is possibly the only means by which a diagnosis can be made. If a mitral murmur ensues in old people in whom there has been physical cause for the development of dilatation and hypertrophy, as in emphysema or arteriosclerosis, it is usually due to relative incompetency of the valve. It must not be forgotten that the mitral area is the seat of a number of murmurs due to various causes. (See Auscultation, page 432.)

Mitral Stenosis. Obstruction to the flow of blood from the auricle to the ventricle is due to valvulitis, or endocarditis, and particularly the endocarditis of early life. It is of much more frequent occurrence in women than aortic disease. It is much more often seen in young adults and children, because its ætiological factors, rheumatism and chorea, are then more prevalent.

On account of the obstruction at the orifice changes ensue in the auricle. These changes depend in a measure upon the nature of the lesion. In the so-called buttonhole contraction they are very marked. The orifice may be so small in rare cases as to admit only a small probe. Dilatation and hypertrophy of the left auricle ensue if the valve-changes take place gradually. The walls of the auricle are thickened to three or four times their natural size. On account of the dilatation of this auricle the outflow from the pulmonary veins is impeded, which in turn obstructs the circulation of blood through the lungs. As a consequence, dilatation and hypertrophy of the right ventricle occur. As a result of this we have, later on, the occurrence of relative incompetency at the tricuspid orifice with engorgement of the systemic veins. The left ventricle does not take part in any changes. It retains its normal size, but it may look small in comparison with the right ventricle.

Symptoms. If hypertrophy of the right ventricle ensues, the compensation may be sufficient to prevent the occurrence of symptoms for many years. The disease may exist for a number of years without discomfort to the patient. Because of its rheumatic origin a fresh endocarditis may develop, particularly as most of the subjects are young. The old valve-lesion invites infection, and so a recurrent form of endocarditis is induced. If fresh endocarditis occurs, embolic symptoms are likely to follow. Embolism takes place particularly in the brain, causing hemiplegia or aphasia. When failure of compensation takes place, the symptoms

described in mitral incompetency arise. They are the symptoms of dilatation of the heart, and may recur frequently during a long period of years.

Dropsy, however, is not so common as in mitral regurgitation. Visceral stases are common when compensation fails, and in many cases we find enlargement of the liver continuing for a long period. Ascites may in rare cases be the only manifestation of mitral obstruction.

Physical Signs. (Plate XXXII., Fig. 2.) The physical signs of mitral obstruction are more striking and more diagnostic of the lesion than the physical signs of any other form of organic heart disease.

INSPECTION. As the disease develops in children with soft ribs the local deformities are marked. For the same reason præcordial bulging is more prominent. Hypertrophy of the right ventricle causes the sternum and the fourth, fifth, and sixth costal cartilages to protrude. The apex-impulse is not usually displaced, certainly not beyond the mid-clavicular line. The impulse is not marked at the apex. In the third and fourth interspaces a visible impulse is seen along the margin of the sternum. After dilatation the extent of impulse diminishes and the veins of the neck become engorged, the blood regurgitating into them during the systole.

PALPATION. In the large majority of cases a distinct fremitus or thrill is felt—more marked in the fourth or fifth interspace, inside of the nipple-line. It is diastolic or presystolic in time and is usually localized to a small area; is increased during expiration, and is of a twisting, grating, or grinding character. It is made up of a series of small shocks increasing in intensity, culminating in a sudden, sharp shock which occurs at the time of the impulse. The presystolic thrill and systolic shock are pathognomonic, and may be present when other signs, as the murmur, are absent or indistinct. The cardiac impulse is felt strongest at the lower margin of the sternum and in the third and fourth interspaces, in some cases even in the second. It is due to an enlarged and dilated right ventricle.

THE PULSE. With perfect compensation the pulse is slow, regular, and of good tension, although small. If the mitral orifice is much narrowed, the pulse is small, weak, and irregular in force and rhythm. When compensation fails and the right heart is dilated, the pulse becomes rapid, quick, weak, small in size, and irregular in force and rhythm. The dilatation may be so great that the right auricle and over-distended veins may press upon the aorta or the innominate and subclavian arteries. The pulse on that side will be lessened in volume.¹

PERCUSSION. The area of cardiac dulness is increased upward and to the right and left of the margin of the sternum. Sometimes it extends upward as high as the second rib; this increase is quite characteristic.

AUSCULTATION. At the apex, or just inside the position of the apex-beat, a murmur is heard, its point of maximum intensity distinctly localized to this spot. It is usually not transmitted. (See Fig. 126.) It is of a churning and grinding character, or vibratory and purring. It is usually high in pitch and rough. It occurs synchronously with the thrill, and terminates with a loud shock that is heard simultaneously with the

¹ Popoff, British Medical Journal, 1893.

first sound. It is therefore *presystolic* in time. As has been said of the thrill, so it may be said of this murmur, that it is the only murmur that is pathognomonic of a special lesion. It indicates narrowing of the mitral orifice. The only exception in which the lesion is absent, although the murmur is present, is found in the class of cases described by Flint, referred to in the section on aortic regurgitation. The first sound is loud, clear, and abrupt; it may be thumping.

The murmur of mitral stenosis may occupy the entire period of the diastole, but in the large majority of cases it occurs in the latter half only, during which the auricular systole occurs. In some instances it is heard in the middle of the diastole.

ASSOCIATE MURMURS. 1. Synchronous with the above adventitious sound a systolic murmur may be heard at the apex, soft and low in pitch, which may be transmitted into the axilla. It is usually due to associated mitral regurgitation. 2. At the lower portion of the sternum a systolic murmur may be heard, due to dilatation and incompetency at the tricuspid orifice. Murmurs in the aortic region are not usually heard.

PRESYSTOLIC MURMUR NOT DUE TO VALVULITIS. A presystolic murmur without mitral obstruction may occur in aortic regurgitation and in adherent pericardium.

The *second sound* at the pulmonary orifice is usually *accentuated*. It is heard in the second and third interspaces along the left edge of the sternum. It may be heard at the apex. *Reduplication* of the first sound is often observed. Reduplication of the second sound is very common. After compensation is broken, other murmurs may be heard, and the presystolic murmur changes in character. It may disappear entirely and be replaced by a sharp first sound. The short, high-pitched systolic shock may continue, although the murmur disappears. It disappears probably because the left auricle has become weakened. The tricuspid murmur continues during this period.

Diagnosis. The points of distinction of mitral obstruction are (1) the position of the murmur; (2) its restricted area; (3) its peculiar character; (4) the systolic shock which takes the place of the first sound; (5) the thrill; (6) the impulse and increased area of dulness upward; (7) accentuated pulmonary second sound; (8) reduplication; (9) the absence of the pulse of aortic regurgitation and of hypertrophy of the left ventricle.

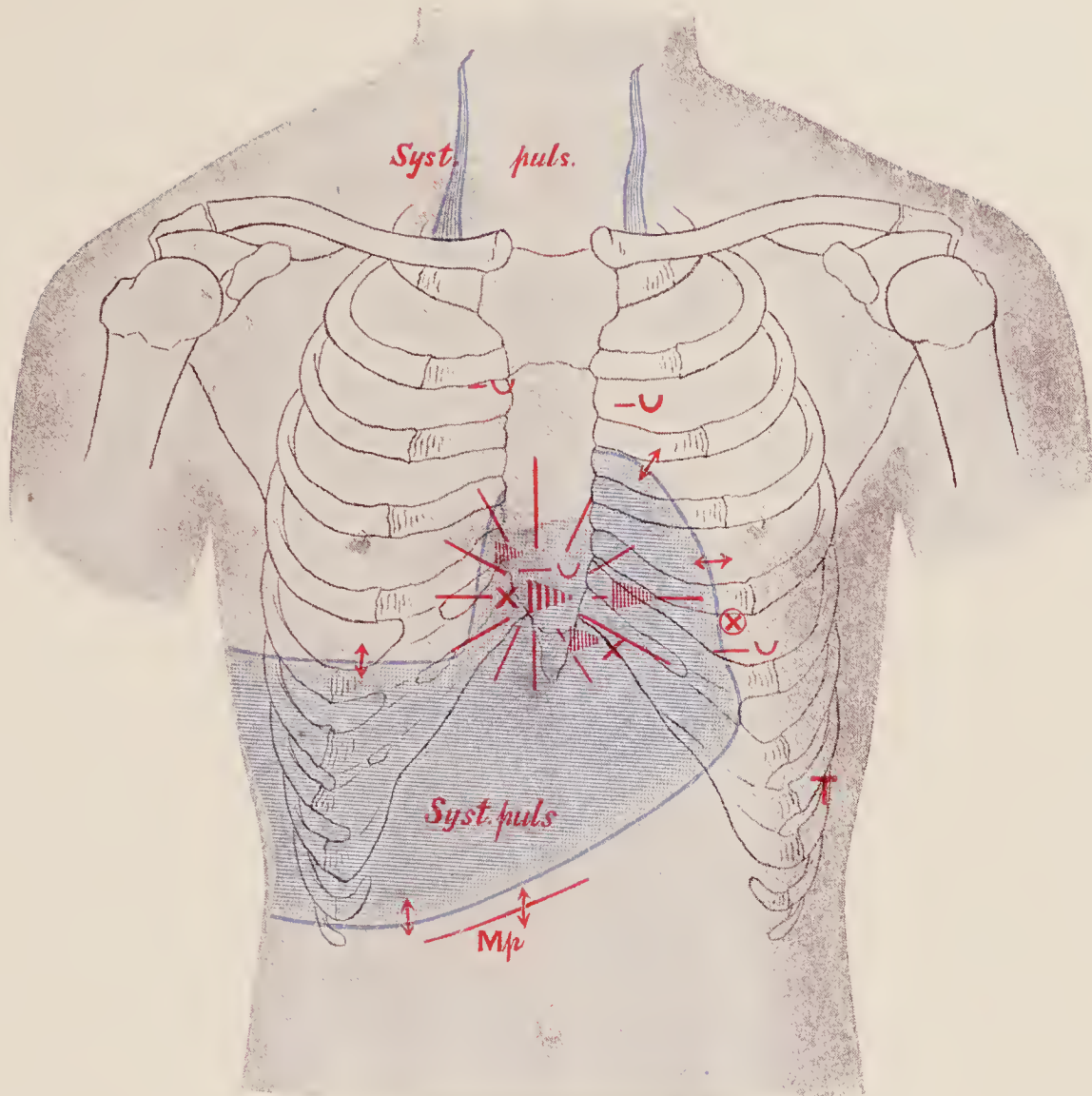
Tricuspid Regurgitation or Incompetence. Structural disease at the tricuspid orifice is of comparatively rare occurrence. Incompetence is more frequent, and is due to dilatation with relative insufficiency of the valve-orifice. It occurs secondarily to obstructive lung diseases, as emphysema and cirrhosis, and is secondary to obstruction and regurgitation at the mitral orifice which lead to stasis in the lungs.

Symptoms. The symptoms were detailed in speaking of the mitral valve affections. They are those of obstruction in the pulmonary circulation and engorgement of the systemic veins.

Physical Signs. (Plate XXXIII., Fig. 1.) **INSPECTION.** The physical signs of dilatation of the right heart are seen. An impulse in the epigastrium is noted. This is seen especially between the xiphoid cartilage

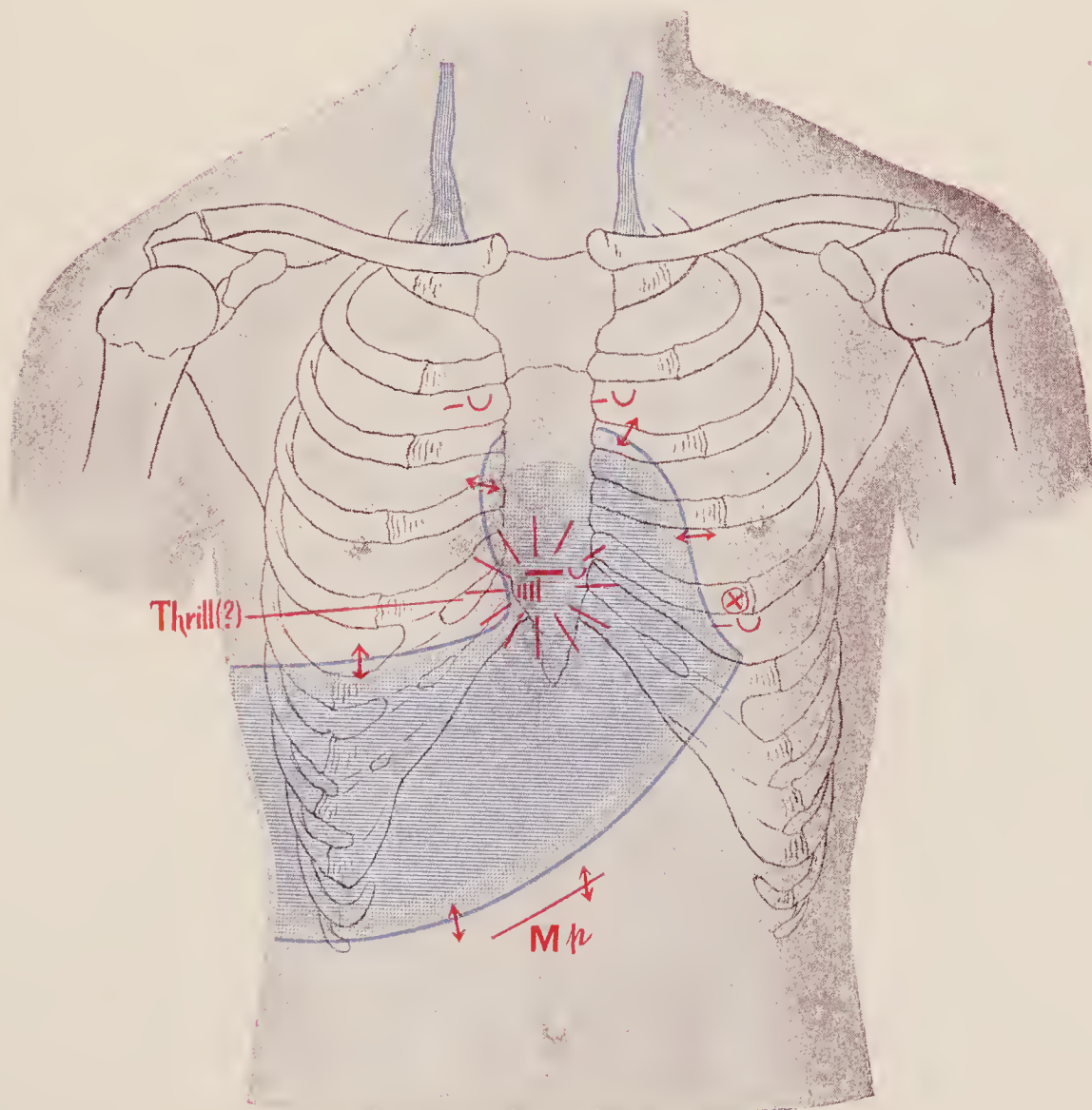
PLATE XXXIII.

FIG. 1.



Tricuspid Regurgitation.

FIG. 2.



Tricuspid Stenosis.

and the left margin of the ribs. Pulsation to the right of the sternum and in the second and third intercostal spaces may also be observed. The veins of the neck are also seen to pulsate. In addition to the wavy pulsation, regurgitation of the blood into the right auricle causes transmission of the pulse-wave into the veins. The pulsation is systolic in time. It is more marked in the right jugular than in the left, and in the external than in the internal veins. With the pulsation, regurgitation is readily observed by emptying the external jugular vein. Place the finger firmly on the vein just above the clavicle, move it along the course of the vein in the direction of the inferior maxillary bone. The vein is thus emptied of blood, and with each systole of the heart it will be seen to fill from below in rhythmical pulsation. The veins are increased in size. This is more noticeable during the act of coughing or when the patient holds his breath in full inspiration. In rare instances the pulsation is transmitted to the subclavian and axillary veins.

PALPATION. By palpation the above conditions are also determined. The impulse over the lower sternum and in the epigastrium is noted to be forcible. The regurgitant pulsation is transmitted to the descending vena cava as well as to the ascending. The hepatic veins also distend during the systole. So-called *pulsation of the liver* is produced. With one hand on the fifth and sixth costal cartilages and the other over the liver in the axillary region, rhythmical expansile pulsation may be recognized. It is not of common occurrence, but is absolutely diagnostic of regurgitation at the tricuspid orifice. It must not be confounded with a pulsation transmitted to the liver from the heart or the aorta.

PERCUSSION. The area of cardiac dulness is increased transversely and upward, as described in mitral stenosis. It extends often far beyond the right edge of the sternum.

AUSCULTATION. A murmur is heard at the xiphoid cartilage, the lower end of the sternum, or the head of the fourth rib. It is *systolic* in time, usually low in pitch, and is heard clearly to the left of the sternum, within an inch of the apex, and to the right of the sternum and the outer limits of percussion-dulness. (See Fig. 124.) It is not transmitted further. Other murmurs, due to the primary organic disease, are heard. If the heart is weak, the lesion may not be productive of a murmur. The pulmonary second sound is accentuated.

Tricuspid Stenosis. Stenosis at this valve-orifice is generally of congenital origin. In rare instances it may be secondary to lesions in the left heart. It is accompanied by dilatation of the right auricle.

Physical Signs. The physical signs (Plate XXXIII., Fig. 2) are the same as in stenosis at the mitral orifice, except for the alteration in their position. In some instances a presystolic thrill has been observed, and with it a presystolic murmur at the lower end of the sternum or toward the right of it. The area of dulness is increased as in right-sided dilatation. Cyanosis is a prominent symptom and may be intense.

Diseases of the pulmonary valve are extremely rare and are almost always congenital.

Pulmonary Insufficiency. (Plate XXXIV., Fig. 1.) The *physical signs* are due to regurgitation into the right ventricle. The maximum intensity is in the second pulmonary interspace, and the murmur is transmitted down the sternum. It can not be distinguished from aortic regurgitation, except by the pulse.

Pulmonary Stenosis. (Plate XXXIV., Fig. 2.) In stenosis of the pulmonary valve a systolic murmur and thrill are detected to the left of the sternum in the second interspace. The murmur is not transmitted to the vessels of the neck. The pulmonary second sound is weak. The effect on the heart is the production of right-sided hypertrophy.

Combined Valvular Lesions. (Plate XXXV.)

It must not be forgotten that there may be disease causing both obstruction and regurgitation at the same time and at the same orifice, or that two or more valves may be the seat of disease in the same individual. It is not impossible, for instance, to have aortic obstruction and regurgitation, mitral obstruction and regurgitation, and tricuspid regurgitation. Aortic obstruction or insufficiency is frequently combined with mitral insufficiency. Aortic and mitral insufficiency occur together most frequently in children; aortic obstruction and mitral obstruction in adults.

When more than one valve is diseased, the site of the various lesions is determined by the time, the position of maximum intensity, and the direction of transmission of the murmurs. Students often experience difficulty here. A systolic murmur may be heard in the aortic area and in the mitral area at the same time. If it is observed that each progressively weakens as the stethoscope is moved toward the middle of the præcordial area, it may be inferred that the systolic murmur is due to two lesions. As previously intimated, the direction of the transmission of the murmur further aids in the diagnosis.

ENLARGEMENT OF THE HEART.

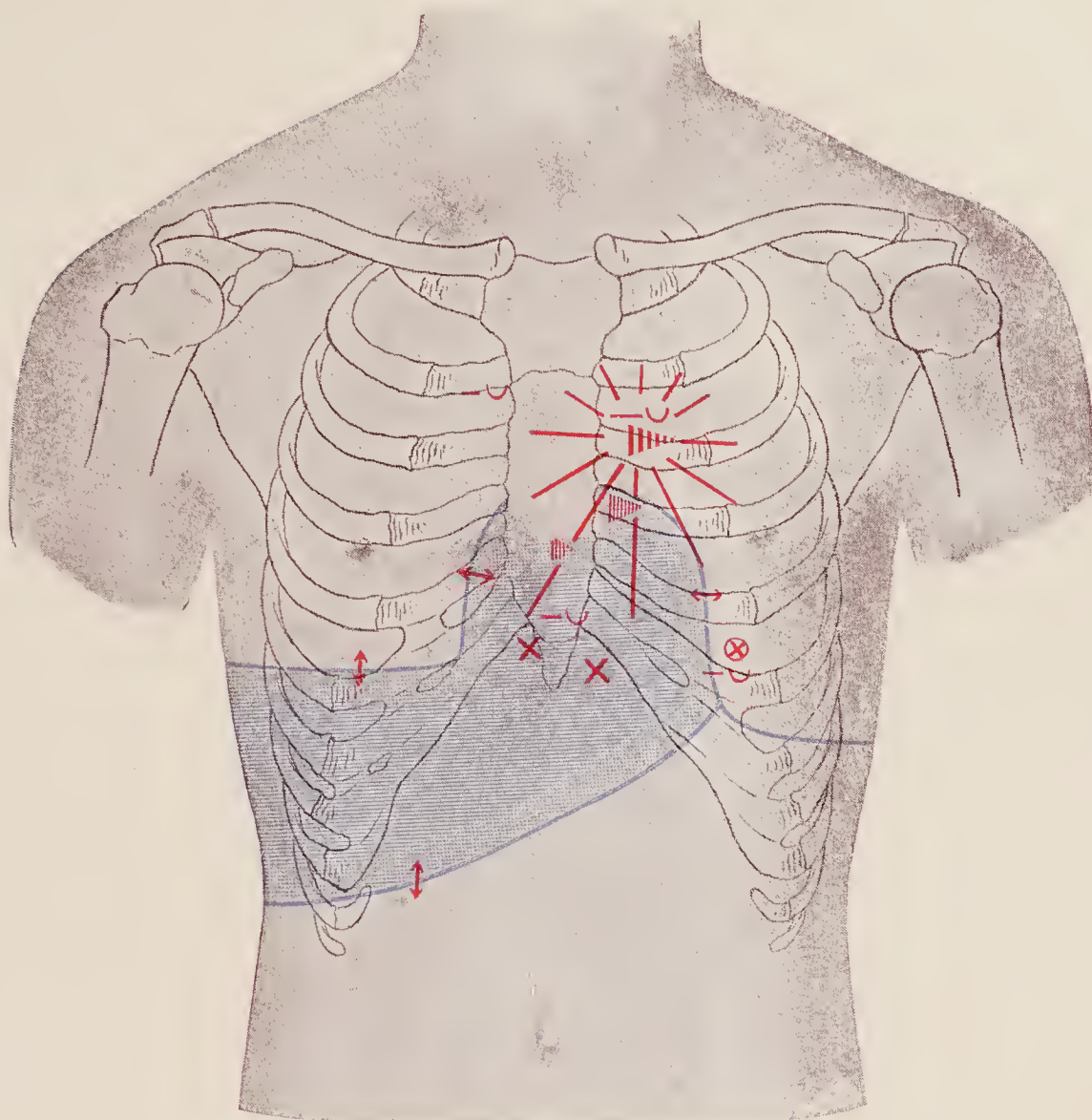
Enlargement of the heart is due to *hypertrophy* or to *dilatation*. In hypertrophy there is increased thickness of the muscular walls. This may be general or limited to the walls of one chamber. Hypertrophy is further divided into simple hypertrophy, in which the cavity or cavities are of normal size, and eccentric hypertrophy, in which, with increase in the wall, there is enlargement of the cavities. This is hypertrophy with dilatation. The left ventricle is most frequently the seat of hypertrophy when one chamber is involved. The cause of hypertrophy is obstruction to the flow of blood; increased work is followed by increased size of the muscle.

General hypertrophy occurs from over-action, from diseases of the heart itself, or from affections of the bloodvessels.

A. *Diseases of the Heart.* 1. Disease of the aortic valves. 2. Pericardial adhesions. 3. Myocarditis of the fibrous variety. 4. Neuroses with over-action and frequent palpitation, as in exophthalmic goitre and

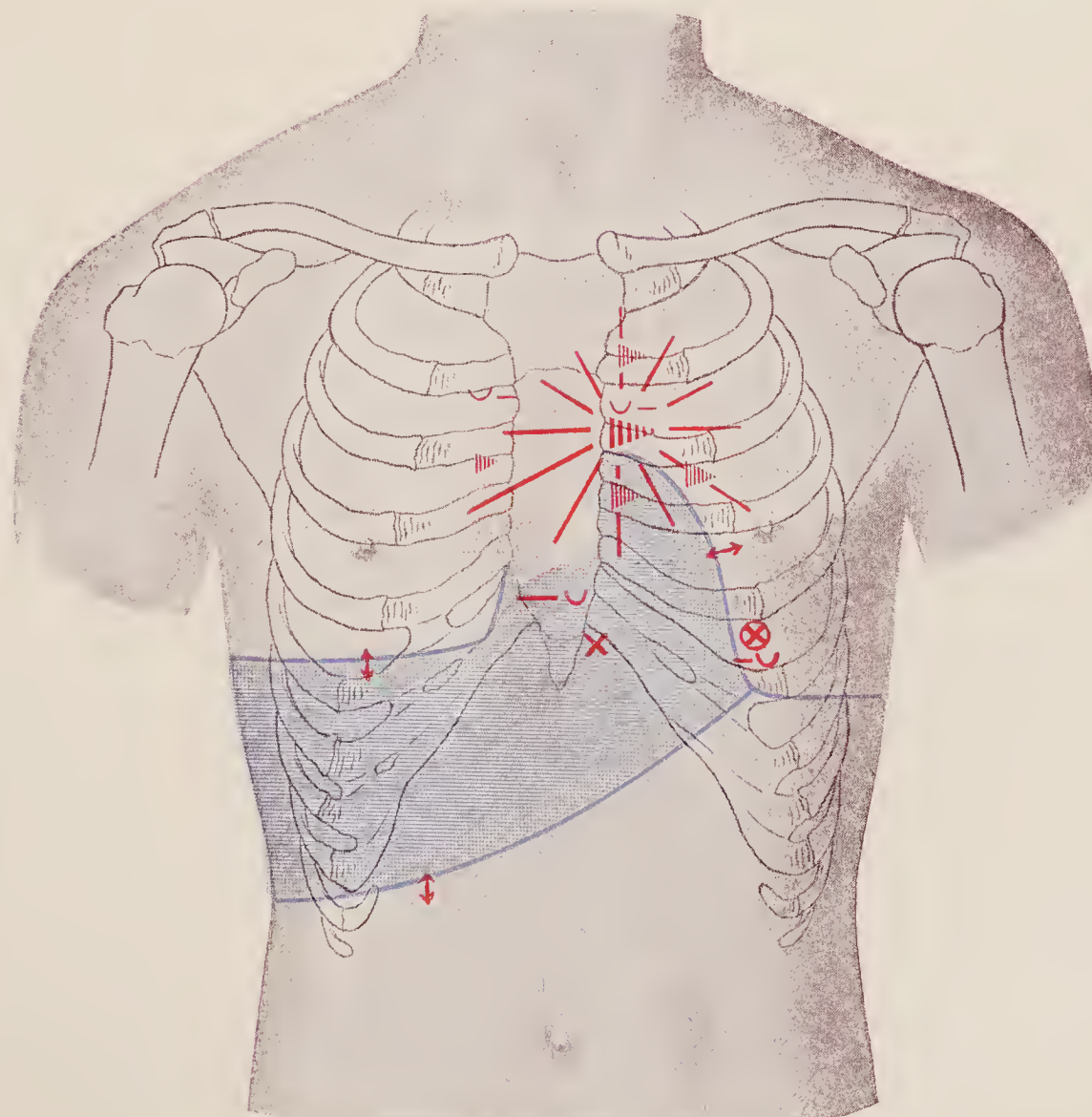
PLATE XXXIV.

FIG. 1.



Pulmonary Insufficiency.

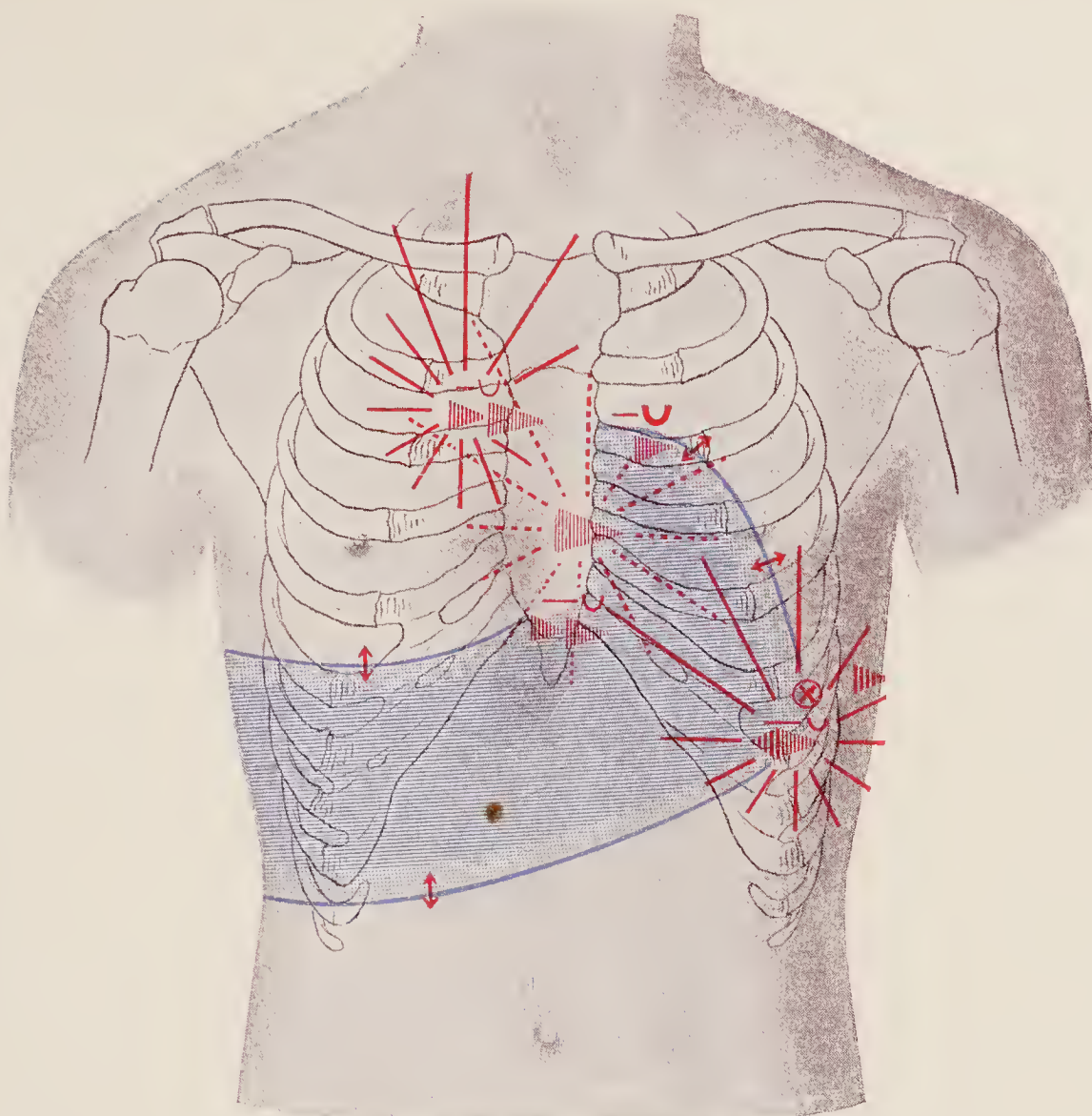
FIG. 2.



Pulmonary Stenosis.

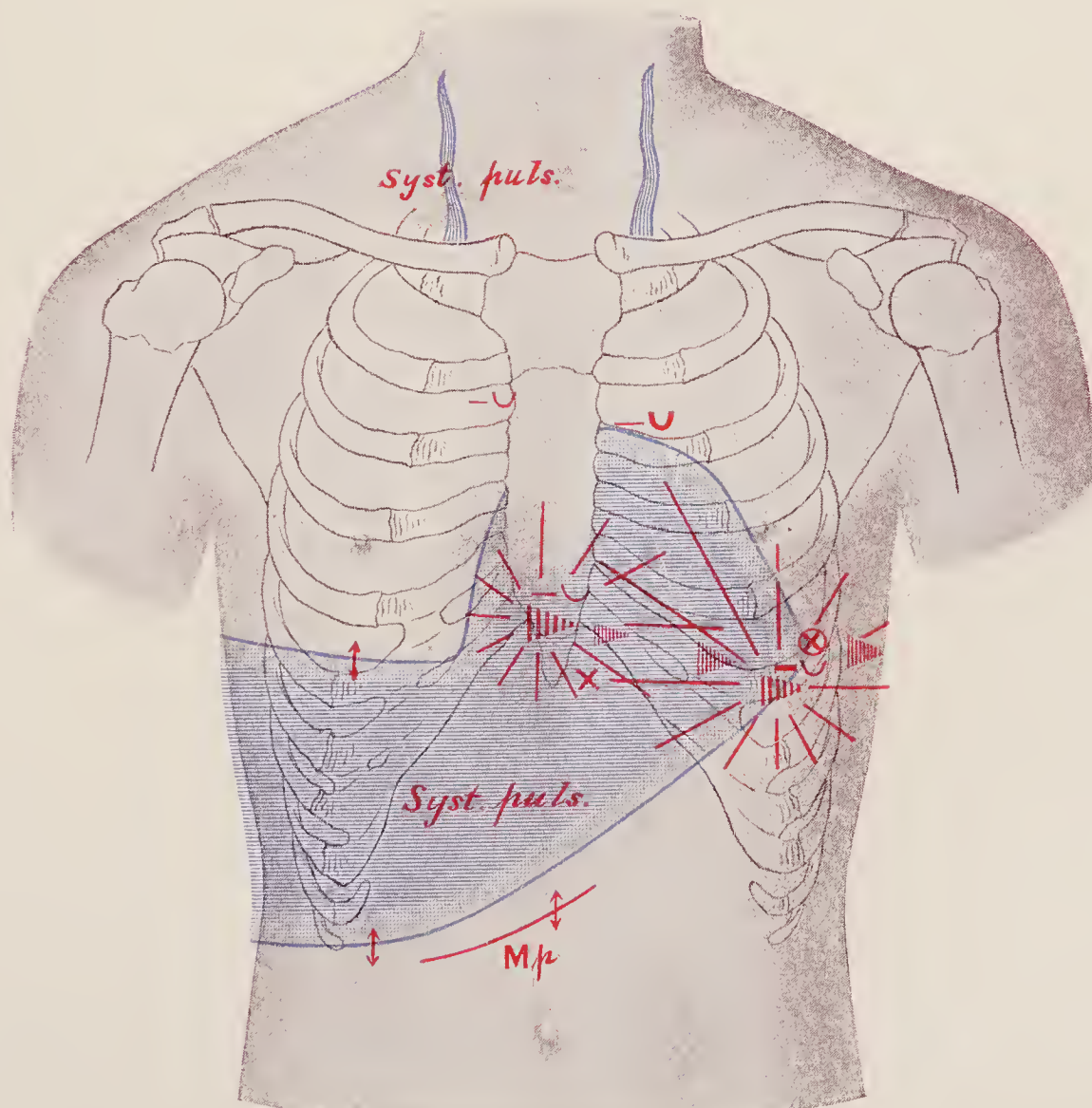
PLATE XXXV.

FIG. 1.



Combined Mitral and Aortic Insufficiency and Stenosis.

FIG. 2.



Combined Mitral and Tricuspid Insufficiency.



PLATE XXXVI.

FIG. 1.

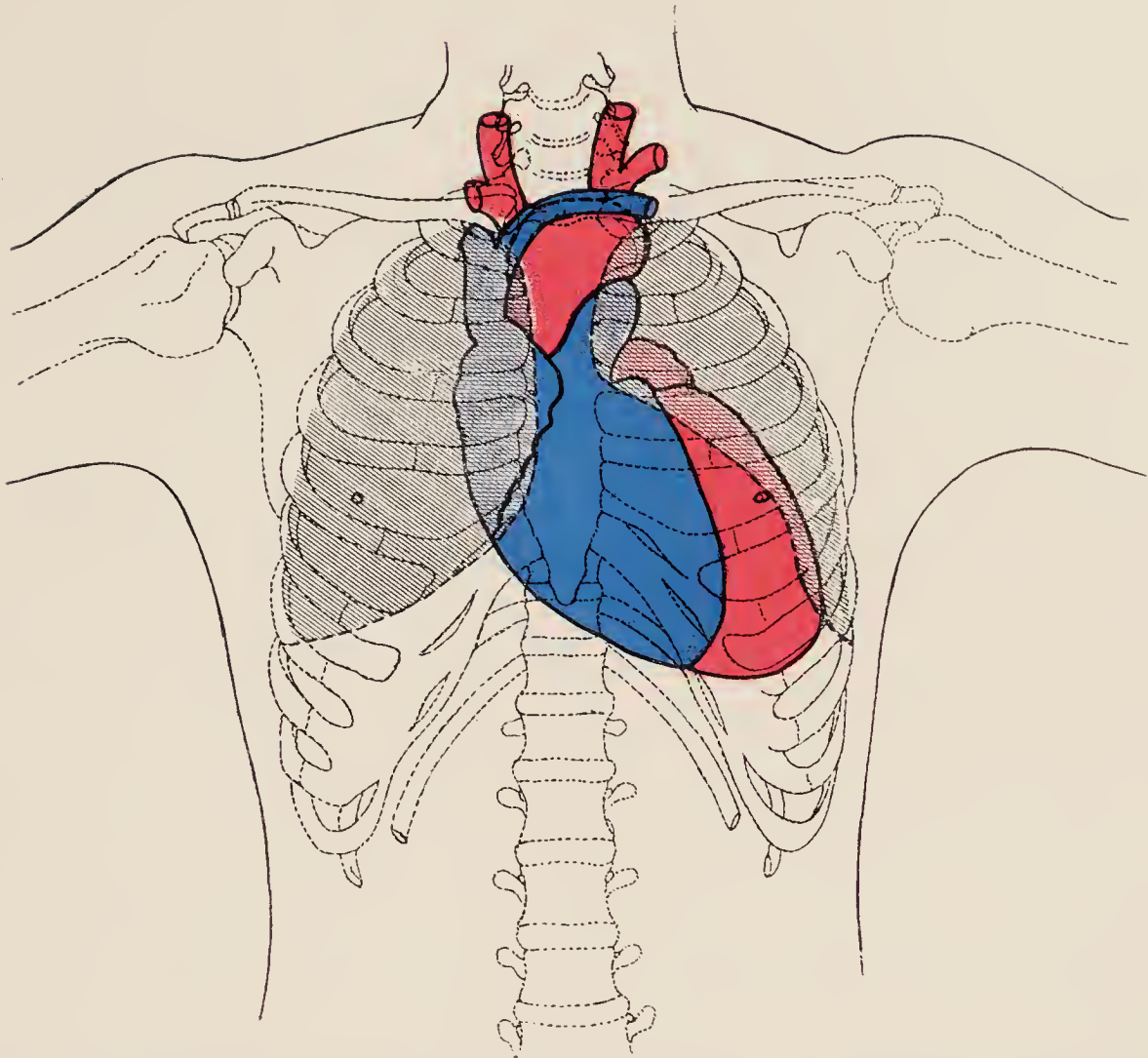


Diagram to illustrate the size and position of the heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness in aortic insufficiency. RED = the left side of the heart (marked hypertrophy and dilatation). BLUE = the right side. The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.

FIG. 2.

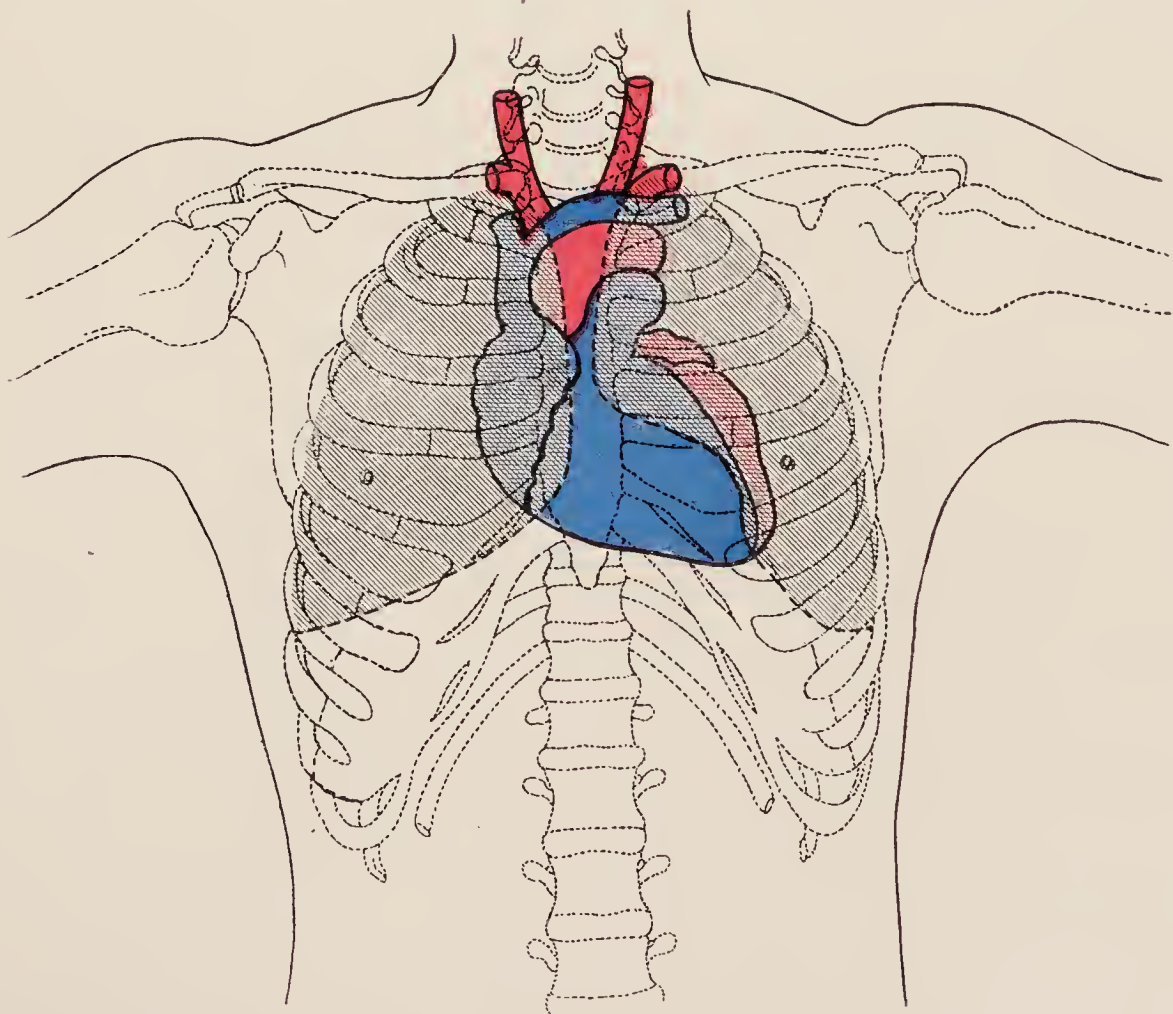


Diagram to illustrate the size and position of the normal heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness. RED = the left side of the heart. BLUE = the right side. The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.

PLATE XXXVI-a.

FIG. 1.

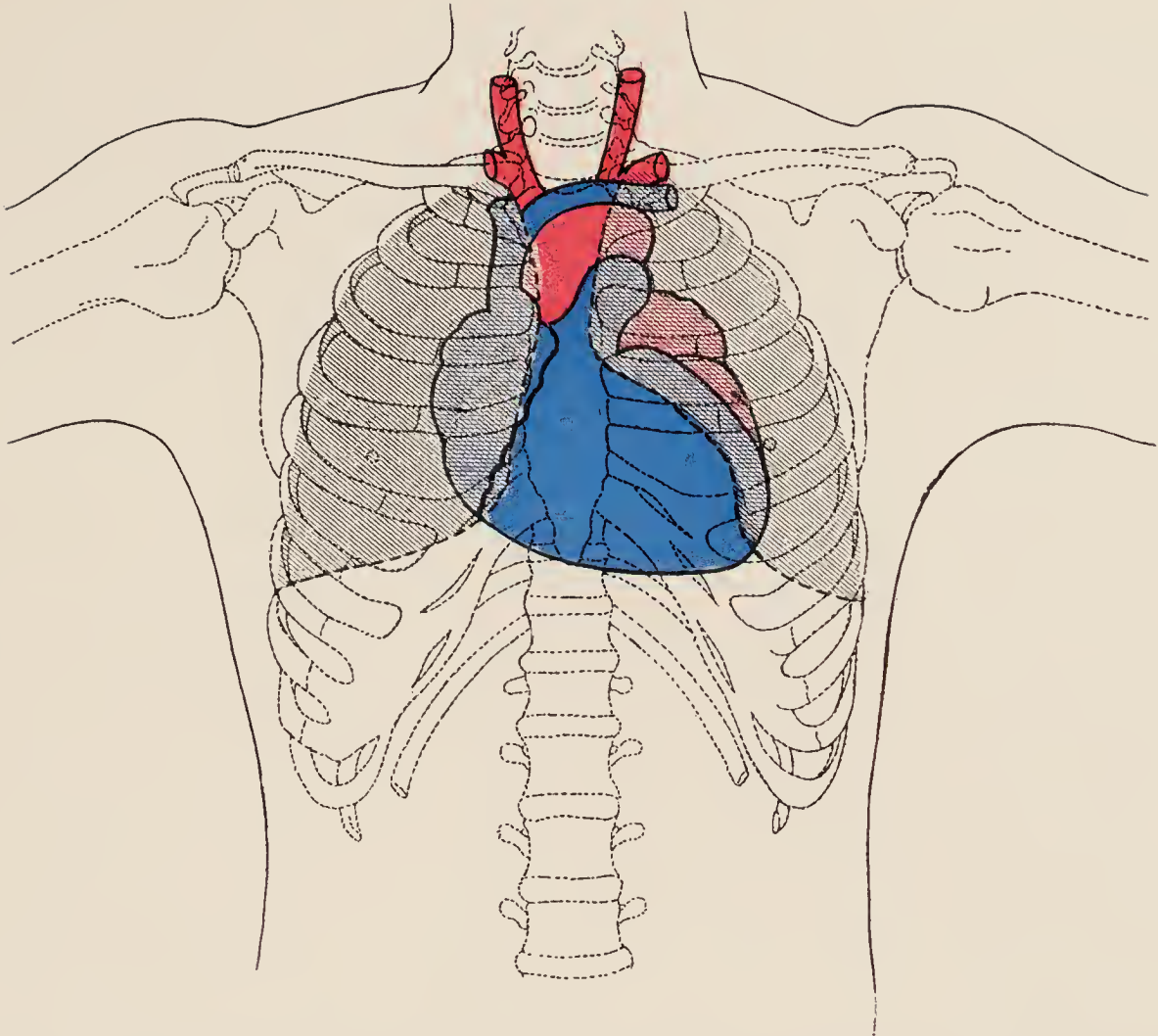


Diagram to illustrate the size and position of the heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness in mitral stenosis. RED = the left side of the heart (hypertrophy and dilatation of the auricle). BLUE = the right side (hypertrophy). The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.

FIG. 2.

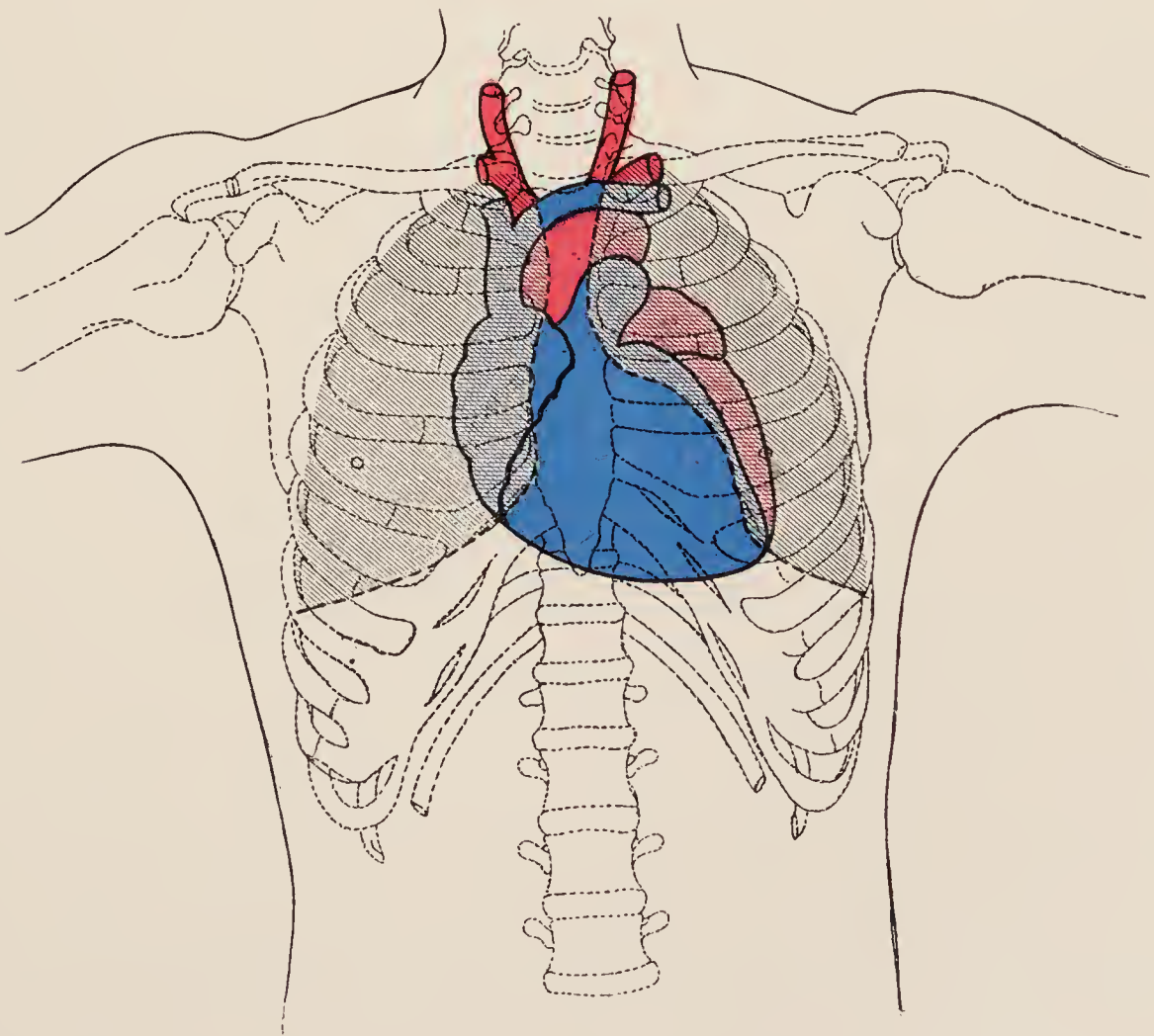


Diagram to illustrate the size and position of the heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness in mitral insufficiency. RED = the left side of the heart (hypertrophy and dilatation). BLUE = the right side (hypertrophy). The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.

from the effects of tea, tobacco, and alcohol. In pericardial adhesions and myocarditis hypertrophy arises because of the inability of the heart to do the work expected of it. There is no obstruction in the course of the vessels or at the orifices. The struggle to keep up causes the hypertrophy. In neuroses there is absence of obstruction, but the rapid action causes hypertrophy. (See Plates XXXVI. and XXXVI.-A.)

B. *Affections of the bloodvessels* which cause hypertrophy are: 1. General arterial sclerosis. 2. Increased arterial tension due to contraction of the peripheral arteries, as in Bright's disease, and in toxæmias from lead, the poison of gout and of syphilis. 3. Increased blood-pressure from prolonged muscular exertion. 4. Narrowing of the aorta from external pressure and from congenital stenosis or the development of an aneurism.

Hypertrophy of the Left Ventricle. Disease of the aortic valves, mitral valvulitis with regurgitation, and those diseases of the vessels which cause general hypertrophy also cause left ventricle hypertrophy.

Hypertrophy of the Right Ventricle. Obstruction to the flow of blood in the pulmonary area is the usual cause of hypertrophy of the right ventricle. This obstruction occurs in lesions of the mitral valve, causing pulmonary stenosis; and in disease of the lungs causing compression of the bloodvessels, as in emphysema or cirrhosis. It occurs if there is disease of the right heart with obstruction of the valves. Thus, in obstruction at the pulmonary orifice the right ventricle undergoes secondary hypertrophy.

Hypertrophy of the Auricles. Simple hypertrophy of the left auricle with dilatation develops in mitral stenosis. Hypertrophy of the right auricle occurs in tricuspid obstruction and in right-sided dilatation with tricuspid regurgitation.

SYMPTOMS. General Hypertrophy. The symptoms of hypertrophy of the heart are *general* and *local*. The former are not common. They are due to increased tension in the cerebral vessels because of increased force of the heart, usually causing congestive headaches, noises in the ears, flashes of light, and flushing of the face.

General symptoms arise in hypertrophy of the left ventricle because the increased force causes reactive spasm of peripheral vessels, and hence increased tension in the vascular system. In Bright's disease, for instance, or heightened arterial tension from other causes, endarteritis develops in the large vessels on account of the strain put upon them. This is seen particularly in the aorta and its divisions. Whether atheroma is primary or secondary, its presence, with hypertrophy of the left ventricle, indicates that rupture of the vessel somewhere in the periphery may take place. This occurs most frequently in the brain, causing apoplexy.

Locally, the patient complains of fulness and discomfort, particularly marked when lying on the left side. In the hypertrophy that accompanies the tobacco-heart, or the irritable heart of soldiers, there may be some pain. On the other hand, the organ may be enormously enlarged without the patient complaining of discomfort about the heart. Palpitation is not of common occurrence except in neurasthenic subjects.

PHYSICAL SIGNS. The hypertrophy causes præcordial bulging, if it has

developed early in life, when the ribs are soft. The intercostal spaces are widened and the area of impulse is much increased. The normal impulse is changed in position. It is downward and to the left, often extending as far as the axilla in hypertrophy of the left ventricle.

Palpation. The impulse is forcible and heaving. The observer's head is visibly raised with each systole when placed upon the chest for auscultation. The impulse is slow. This slow, heaving impulse distinguishes it from the forcible impulse of dilated hypertrophy, which is sudden and abrupt. Inspection is confirmed as to the position of the apex. In moderate hypertrophy the apex extends to the sixth interspace in the midclavicular line. In extreme hypertrophy it may extend to the seventh interspace. The heart may be apparently hypertrophied in fibrous and fatty myocarditis; on the other hand, the impulse may be absent in emphysema, in fatty over-growth of the heart, and in persons with thick chest-walls.

The Pulse. The frequency of the pulse is not affected. It is full, regular, and strong. The tension is increased. In dilated hypertrophy the pulse is full but soft, and more rapid than in simple hypertrophy. When failure of the heart takes place, the pulse increases in frequency and becomes intermittent and irregular. When valve-lesions are present, the pulse is modified accordingly.

Percussion. The area of dulness is increased both upward and transversely. It may begin as high as the second interspace and extend two inches beyond the left midclavicular line, and an inch beyond the right edge of the sternum transversely. In simple hypertrophy the area is ovoid.

Auscultation. When the valves are healthy, prolongation of the first sounds occurs. They are also at times duller than in health. The dull, prolonged first sounds distinguish hypertrophy from dilatation, for in the latter they are clear and sharp. The second sounds are clear and loud. The degree of accentuation depends upon the state of the peripheral arteries. If there is heightened tension, the second sound may be reduplicated. If valvular disease is present, the sounds are modified.

Hypertrophy of the Left Ventricle. The symptoms of general hypertrophy obtain in left ventricle hypertrophy.

Hypertrophy of the Right Ventricle. Increased pulmonary tension from resistance in the pulmonary circulation may always be looked for in this condition. If there is complete compensation, no symptoms are observed, or only dyspnœa on unusual exertion. Hypertrophy of this ventricle persists for a long period without the grave local changes in the heart, or secondary changes in the peripheral vessels that occur in left ventricle hypertrophy. In dilated hypertrophy, when the dilatation is in excess, tricuspid regurgitation takes place, with the development of venous stases. Induration of the lungs may succeed the persistent engorgement of the capillaries. Pulmonary congestions and apoplexy may also occur.

PHYSICAL SIGNS. The physical signs of hypertrophy of the right ventricle have been referred to under the various valve affections. There is bulging of the lower part of the sternum and cartilages. The epigastric impulse in the angle between the ensiform cartilage and the ribs has been

referred to. The impulse may be in the sixth interspace. It is diffuse ; it may extend upward as in mitral stenosis. Cardiac dulness is increased toward the right an inch or more beyond the border of the sternum. The heart-sounds are not much changed unless there is dilatation. The tricuspid sound is clear and sharp when this occurs. The pulmonary second sound is accentuated, and reduplication may take place. The radial pulse is small. If there is tricuspid regurgitation, the physical signs that attend it are present.

Hypertrophy of the Left Auricle. This is present in mitral stenosis, but can not be determined by physical signs, save possibly by greater increase of dulness to the left of the sternum in the second and third interspaces. Barr states that dulness above the "suprasternal-mammillary line" toward the left clavicle indicates enlargement of the left auricle, as in mitral stenosis. The line above mentioned is drawn from the middle of the suprasternal notch to the normal site of the left nipple on the fourth rib.

Hypertrophy of the Right Auricle. Hypertrophy of the right auricle with dilatation occurs under the same circumstances as hypertrophy of the ventricle. The right usually dilates more than the left auricle in left ventricle hypertrophy. There is increased area of dulness in the third and fourth right interspaces ; abnormal pulsation is sometimes observed in this situation before the systole, with the signs of tricuspid regurgitation.

DIAGNOSIS. The forcible impulse in nervous palpitation of the heart must not be confounded with true hypertrophy, although it must not be forgotten that hypertrophy frequently follows neurotic palpitation, as in the smoker's heart, or in exophthalmic goitre. The enlargement must not be confounded with enlargement of the area of cardiac dulness in the præcordial region from other causes, such as pericardial effusion ; aneurism and mediastinal tumor, pushing the heart against the chest-wall ; disease of the lungs, on account of which they are withdrawn from the surface of the heart, as in phthisis or chronic pleurisy ; and displacement of the heart from pressure, as in effusion on the left side of the chest or in disease below the diaphragm. The cause of hypertrophy should be ascertained, for it is a valuable aid in diagnosis. It must not be forgotten that emphysema of the lung may mask a considerable hypertrophy of the heart by causing diminution of the area of dulness.

Dilatation of the Heart. Enlargement due to dilatation of the heart is common. The condition usually succeeds hypertrophy. Thickening of the muscles attends dilatation of the cavities, as in dilated or eccentric hypertrophy. The dilatation occurs because of increased pressure within the cavities or because of weakening of the heart-walls, the pressure within being normal.

1. Increased pressure within the walls is due to an increased amount of blood within the chamber from regurgitation, or from an obstacle to the outward flow of blood. Simple hypertrophy occurs first in many cases ; in others, hypertrophy with dilatation ; in not a few, dilatation takes place at once. In dilatation the chamber does not empty itself during the systole. It is seen physiologically after the exertion of ascending a great height. It may remain within the bounds of physiological action.

The dilatation may be temporary, as is demonstrated after running violently, by increased epigastric pulsation and increased cardiac dulness. The tricuspid valves temporarily become incompetent, owing to their safety-valve action. The latter may continue after the acute strain, the heart always showing symptoms of the condition; or it may disappear entirely. An excessive dilatation results in heart-strain, with cardiac distress and dyspnœa, symptoms due to over-distention and paralysis of the heart. (See Symptoms.) Dilatation occurs in all forms of heart-lesions previously described. The most typical form is seen in aortic regurgitation when the left ventricle is affected and in mitral regurgitation when the left auricle becomes the seat of dilatation.

2. Disease of the heart-walls, lessening the resisting power, the normal pressure within the cavities being maintained, invites dilatation. Acute dilatation may ensue in myocarditis and in infections. It occurs in scarlatinal dropsy, typhoid fever, rheumatic fever, and erysipelas. Certain changes which the heart muscle undergoes in acute endocarditis and pericarditis may lead to dilatation. In anæmia and chlorosis the same process may take place. In chronic myocarditis dilatation takes place at the apex. When pericardial adhesions are present, the fibrous overgrowth invades the interstices of the myocardium, thereby weakening the heart muscle. Dilatation may follow.

Symptoms. The symptoms of dilatation are the reverse of those of hypertrophy. When compensation fails, the blood is not expelled from the chambers in systole, so that the cavity is over-distended with blood which accumulates during the diastole. Weakening of the muscles also favors the development of dilatation. As soon as dilatation becomes permanent, incompetence of the valves takes place. In aortic obstruction endarteritis the left side is first affected. It may be compensated for by hypertrophy of the right side. When this fails, venous engorgement and dropsy ensue. The symptoms have been described under chronic valvular disease. In *acute dilatation* there is a sudden occurrence of dyspnœa, and pain, or at least præcordial oppression may be complained of. The heart's action increases in frequency. The pulse is rapid, feeble, irregular, and may be barely palpable at the wrist.

Physical Signs. **INSPECTION.** The apex is displaced to the left, even so far as the axillary line, but rarely downward unless the hypertrophy precedes the dilatation. The impulse is diffused and undulatory in appearance. The apex-beat is defined with extreme difficulty. It may be visible when the patient leans forward, yet not felt.

With the diffuse area of impulse a quick apex-beat may be felt—much weakened, however. When the right ventricle is dilated, the impulse is seen and felt to the right or left of the xiphoid cartilage, and there is a wavy pulsation along the left edge of the sternum in the fourth, fifth, and sixth interspaces. If the dilatation is extreme, involving the right auricle, a pulsation at the third right interspace close to the sternum may be felt. Tricuspid regurgitation is then present.

The area of dulness is increased in the same directions as in hypertrophy, if the two coexist. In general, it may be said that the increase extends outward to the right or left, the direction corresponding to the

ventricle affected. It is increased upward along the left edge of the sternum in left auricle dilatation. (See Mitral Valvulitis.) When the whole heart is dilated, the increase of dulness is in a transverse direction on both sides. The apex is rounded or square, not pointed as in hypertrophy; indeed, it retains the oval shape of the dulness of a normal heart. As dilatation occurs so frequently in emphysema of the lungs, the modification of the percussion-sound must be remembered.

AUSCULTATION. The systolic sounds are short and sharp. They are high-pitched and resemble the diastolic. The latter may become enfeebled when the dilatation becomes excessive. The right and left first sounds may differ somewhat in intensity, and reduplication may occur. The sounds may be obscured by murmurs. The murmurs are due to previous valve disease or to incompetency on account of dilatation. The action of the heart is irregular and intermittent. The pulse is correspondingly small. In dilatation the alteration of the rhythm is extreme. There may be *embryocardia* or foetal heart rhythm, in which the first and second sounds are alike and the long pause is shortened. More frequently there is galloping rhythm of the heart. It must not be forgotten that, as dilatation ensues, murmurs of various valve-lesions may disappear, particularly the murmur of mitral stenosis. On the other hand, in the earlier stages particularly, murmurs develop on account of incompetence at the auriculoventricular orifices, in addition to the primary organic murmur. These murmurs in turn may disappear if the dilatation is controlled by careful treatment.

DISEASES OF THE ARTERIES.

Arterial Sclerosis or Arterio-capillary Fibrosis.

This occurs as the result of the wear and tear of life and as the accompaniment of age. The time of its onset depends upon the quality of the arterial tissue which the individual inherited, and upon the amount of wear and tear. It may occur early in life, and entire families may show this tendency. Very frequently the sclerosis develops from intoxications of the system, on account of which persistent spasm of the small vessels is set up; for blood of an impaired quality is passed with greater difficulty through the capillaries, as was taught by Bright. The blood-tension is raised thereby. Alcohol, lead, and the poisons of gout and syphilis lead to this condition. The poisons of gout and syphilis may set up directly an inflammation and degeneration of the arteries. In renal disease arterial sclerosis is of common occurrence. The relation to the renal lesion differs. It may be primary or secondary. When primary, the morbid cause operates upon the kidneys as well as the arteries. When secondary, a morbid poison is retained within the system by the diseased kidneys, the action of which is such as to cause peripheral spasm and heightened tension.

Over-filling of the bloodvessels from excessive eating and drinking is thought by some to cause arterial sclerosis through constant over-distention of the vessels. In over-work of the vessels and excessive strain there is

either heightened tension or increased peripheral resistance, the effect upon the bloodvessels being the same in either case. The result of the above causes is thickening of the intima, followed by changes in the media and adventitia, terminating in endarteritis deformans of the large arteries.

Symptoms. The symptoms vary. They may be general or local. The disease may be present although the patient dies from other causes. Local symptoms are due to rupture of the vessels, as in apoplexy from cerebral hemorrhage, or to their obstruction, as the coronary artery, or to rupture of an aneurism.

Physical Signs. Arteriosclerosis is recognized by inspection, palpation, and auscultation of the bloodvessels, and by observation of the condition of the heart. The superficial bloodvessels are elongated and tortuous, and pulsate visibly. On palpation the artery feels hard to the touch; it resists compression; it is corded or rounded underneath the finger, and readily rolled about. The pulse shows at once high tension; the wave is slow in ascent, continues long underneath the finger, and subsides slowly. If in the interval of the beats the vessel remains full, the pulse, as previously noted, is obliterated with difficulty. Sphygmographic tracings are characteristic. (See Pulse.) If after pressure on the radial artery it can still be felt beyond the point of compression, its walls are sclerosed; whereas, if after such compression the artery is obliterated beyond the point of compression, the hardness and firmness of the pulse previously observed are due to vascular tension and not to thickened walls. The two conditions should be distinguished. Hypertrophy of the heart occurs early in the course of the sclerosis, on account of peripheral resistance. The hypertrophy involves the left ventricle, and is not attended by dilatation. The apex-beat is beyond the midclavicular line and is usually displaced downward; the impulse is heaving and forcible. The second sound at the aortic cartilage is characteristic. It is clear and ringing; it is heard in the course of the bloodvessels, and is most distinct at or just beyond the apex. Right-sided hypertrophy and dilatation are not generally present. Auscultation of the larger arteries, as the carotids, the abdominal aorta, and femorals, shows a systolic murmur usually rough and high in pitch. All the above-mentioned conditions may be present, and yet the patient remain in apparent good health. The hypertrophy seems to compensate for the arterial occlusion. There may be no renal disease, or moderate renal cirrhosis may be present, indicated by transient albuminuria, polyuria, and hyaline tube-casts. The subsequent symptoms are due largely to closure of one or more vessels in the peripheral circulation, to the development of an aneurism or dilatation of the aorta, to failing hypertrophy of the heart, or to the development of renal cirrhosis.

The blocking of peripheral arteries is due to embolism or thrombosis, more frequently the latter, and to rupture of peripheral vessels, or in all probability miliary aneurisms. When occlusion of the vessels takes place in arteries which supply the extremities, gangrene may occur. Sometimes the occlusion is due to simple narrowing of the vessels alone. Gangrene of the feet is frequently the result of diseased arteries. If the occlusion takes place in the vessels of the brain, various secondary lesions are

produced. Acute and chronic softening occur in more or less general occlusion from sclerosis of the smaller arteries. Hemiplegia, monoplegia, or aphasia may occur—temporarily, if relieved by collateral circulation, or permanently from embolism, thrombosis, or rupture of the vessels. Hence, apoplexy is almost always due to primary disease of the arteries, in the walls of which, in the large majority of cases, miliary aneurisms have existed. If the coronary arteries are blocked, thrombosis with sudden death takes place, or chronic myocarditis may develop with subsequent aneurism and rupture. Angina pectoris, with or without thrombosis of the coronary artery, is always associated with arterial sclerosis.

Failure of the hypertrophied heart leads to dilatation with all the symptoms previously described, including cyanosis, visceral congestions, and dropsies. The murmur at the apex, due to incompetency from dilatation, may simulate chronic valvular disease, although the latter may never have been present. The sclerosis may advance more rapidly in the kidneys than in the other portions of the circulation; later, on account of the contracted kidney, symptoms of interstitial nephritis may arise.

Aneurism.

A true aneurism is formed by the distention of one or more of the arterial coats. It is usually saccular, but may be cylindrical or fusiform. The fusiform and saccular are the forms most commonly seen. False aneurism, or dissecting aneurism, arises from laceration of the internal coat of the artery. The blood dissects between the layers. It occurs in the aorta. It may begin at the heart and separate the coats as far down as the iliac arteries. *Arteriovenous aneurism* is seen when communication between an artery and a vein has been set up. If a sac intervenes, it is called a *varicose aneurism*. Sometimes communication is direct, the vein becoming dilated, tortuous, and pulsating. It is known as an *aneurismal varix*.

An aneurism may occur in the course of arterial sclerosis from diffuse distention of the coats. Its typical form is seen in dilatation of the aorta with one or more sacculated aneurisms on its surface.

Sacculated aneurism occurs from rupture of the tunica media, independently of more extensive disease of the arteries, and in arterial sclerosis. The most common seat is the ascending portion of the aorta. It occurs early in the course of arterial sclerosis. Such form of aneurism is seen in the smaller vessels. Aneurisms also arise after the lodgement of an embolus permanently plugging the vessel, the proximal end of the vessel becoming dilated.

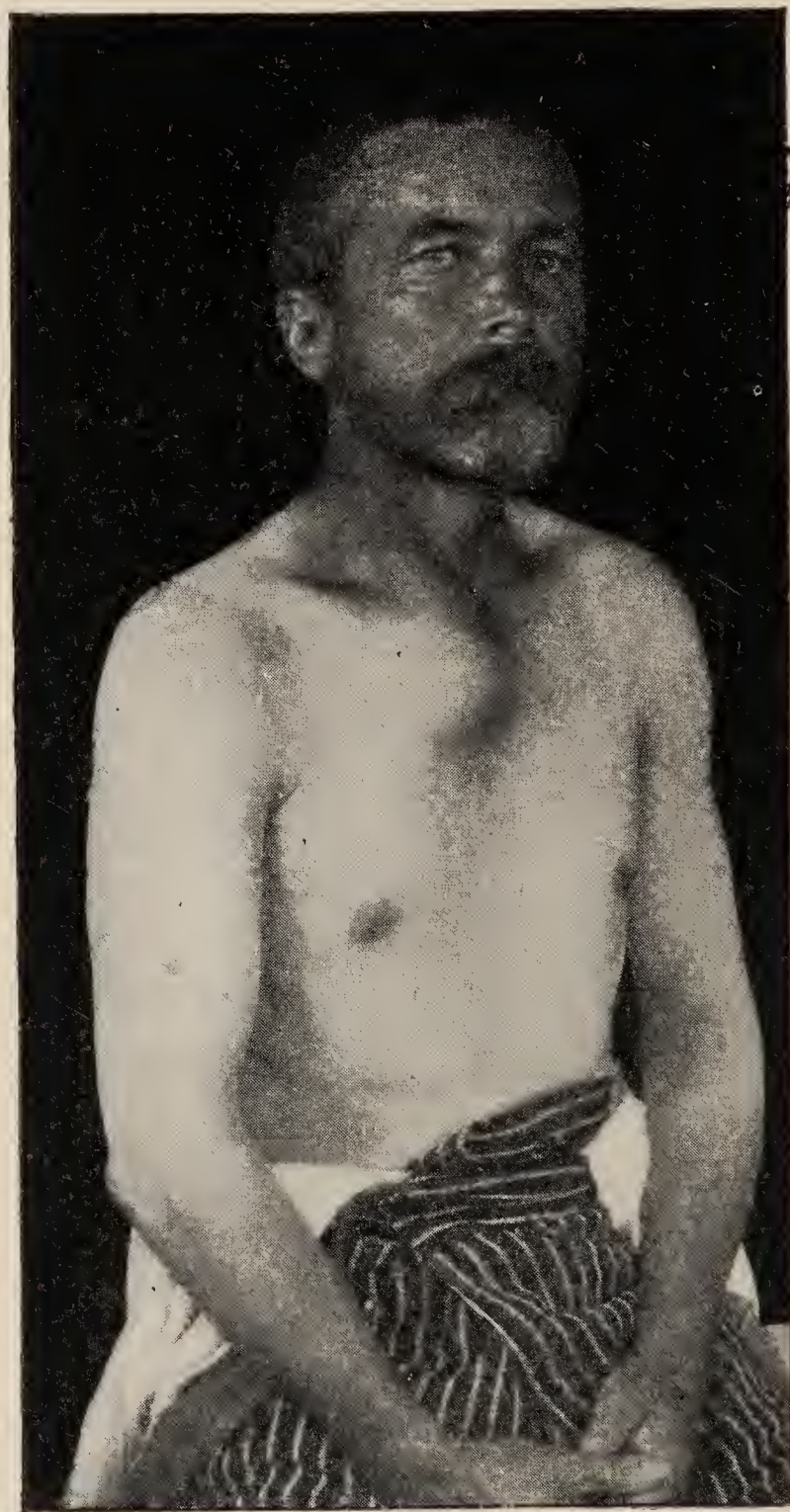
Mycotic aneurism, first described by Osler and exhaustively by Eppinger, occurs in malignant endocarditis. The aneurisms are small in size and multiple, and not recognized during life. They arise from the injury produced by the local infection of bacteria in different portions of the vascular system.

Aneurism of the Thoracic Aorta. The causes which produce arterial sclerosis are operative in the thoracic portion of the aorta—chiefly physical over-work, alcohol, syphilis, and gout. It may be

situated just beyond the aortic ring, at the junction of the ascending and transverse aorta, in the transverse, or at the beginning of the descending portion of the thoracic aorta. The larger aneurisms are at the two bends of the aorta.

Symptoms. The symptoms of aneurism are largely due to pressure, and depend upon the position of the aneurism and the direction of its growth.

FIG. 351.



Aneurism of ascending portion of arch of aorta. Tumor in first and second interspaces, extending into neck. Portion of sternum atrophied. (Original.)

Aneurisms, however, may exist *without symptoms* or *appreciable physical signs*. Even in a patient who has been under careful observation sudden death may take place from rupture of a concealed aneurism, the presence of which had not been suspected during life. On the other hand, cases occur with characteristic pressure-symptoms and with no physical signs.

Aneurisms of the *ascending portion of the arch* cause dislocation of the

heart outward or toward the right pleura or forward. They appear at the second or third right interspace, causing erosion of the ribs and sternum. The vena cava is compressed, causing enlargement of the veins of the head and arms; the subclavian vein may be compressed alone, causing enlargement and œdema of the right arm. Localized œdema may result, confined to the thorax. (See Œdema.) If the aneurism is large, the inferior vena cava may be pressed upon, causing œdema of the feet. The right laryngeal nerve may be involved, causing aphonia and dyspnœa. Pain attends the aneurismal process.

Aneurisms of the *transverse portion of the aorta* project below, forward, or backward. When forward, they produce tumors behind the manubrium, which from pressure cause destruction of the bone; if the aneurism projects backward, marked pressure-symptoms are produced. When the trachea is pressed upon, it causes dyspnœa and cough which is paroxysmal. (See Dyspnœa.) The œsophagus may be pressed upon, causing dysphagia. The left recurrent laryngeal nerve may be pressed upon, causing paralysis of the corresponding vocal cord, with aphonia; or there may arise hoarseness or a peculiar monotonous quality and inability to reach a high note. (See Larynx.) Pressure on a bronchus may produce bronchorrhœa and dilatation, which in turn may lead to localized abscess. The growth may extend upward, involving the coats of the innominate and carotid arteries on the right side, or carotid and subclavian on the left, markedly interfering with the pulse of the two sides. Pressure on the sympathetic nerve is likely to take place in this situation, with contraction of one of the pupils, although at first it is sometimes dilated. The thoracic duct is sometimes compressed, leading to rapid wasting.

In the *descending portion* the pressure-signs of aneurism are often not so marked. The vertebræ are likely to be pressed upon in this situation. The pain, therefore, is most intense. The œsophagus and left bronchus are compressed. Dysphagia and bronchiectasis, the latter causing bronchorrhœa with subsequent gangrene, are likely to occur. The *cough* and the fever in bronchorrhœa, together with emaciation, simulate phthisis, for which aneurism is often mistaken. The physical signs of phthisis are usually pronounced in this situation, and, with the presence of bacilli in the sputum, render the diagnosis easy. In these cases rupture takes place into the bronchus or into the œsophagus. In one of my cases, which had been treated for tuberculosis because of small hemorrhages, with the conditions above mentioned, death took place from rupture into the bronchus, causing sudden profuse hemorrhage. When the aneurism is adherent to the œsophagus and slowly ulcerating into it, rupture may take place, followed by instantaneous death. The vertebræ may be eroded and symptoms of spinal compression arise.

I once saw an autopsy performed by a medico-legal expert on a case of sudden death from gastric hemorrhage. The source of the hemorrhage could not be ascertained. There was blood in the stomach. When he was about to give up the search, the œsophagus and aorta were suggested for examination. A small aneurism was found which had ulcerated and then ruptured into the gullet. In another case the aneurism had ruptured into the pleural sac, causing internal concealed hemorrhage and death.

SPECIAL SYMPTOMS. While pressure-symptoms are the most striking symptoms of this affection, *pain*, which is usually due to pressure, must be referred to. It is an important and a constant symptom. It is sharp and lancinating, and may occur in paroxysms. It is more severe and constant when bone is eroded by pressure on the vertebræ, or on the ribs and sternum. The gnawing pain that attends ulceration of bone subsides after perforation has taken place. Anginal attacks may attend the neuralgic pains just described. Pain sometimes follows the course of the nerves, extending down the arm or to the neck, or along the course of the intercostal nerves.

Cough. The cough is peculiar. It is paroxysmal in many cases and of a brazen, ringing character, indicating its laryngeal origin, due to pressure upon the recurrent laryngeal nerves. It is frequently paroxysmal when the pressure is directed upon the windpipe or bronchus. In the former instance the cough is dry, in the latter tracheal and bronchial. It is attended by a thin, watery expectoration which, if bronchiectasis with fermentation ensues, becomes thick and ropy. *Dyspnœa* occurs more

FIG. 352.

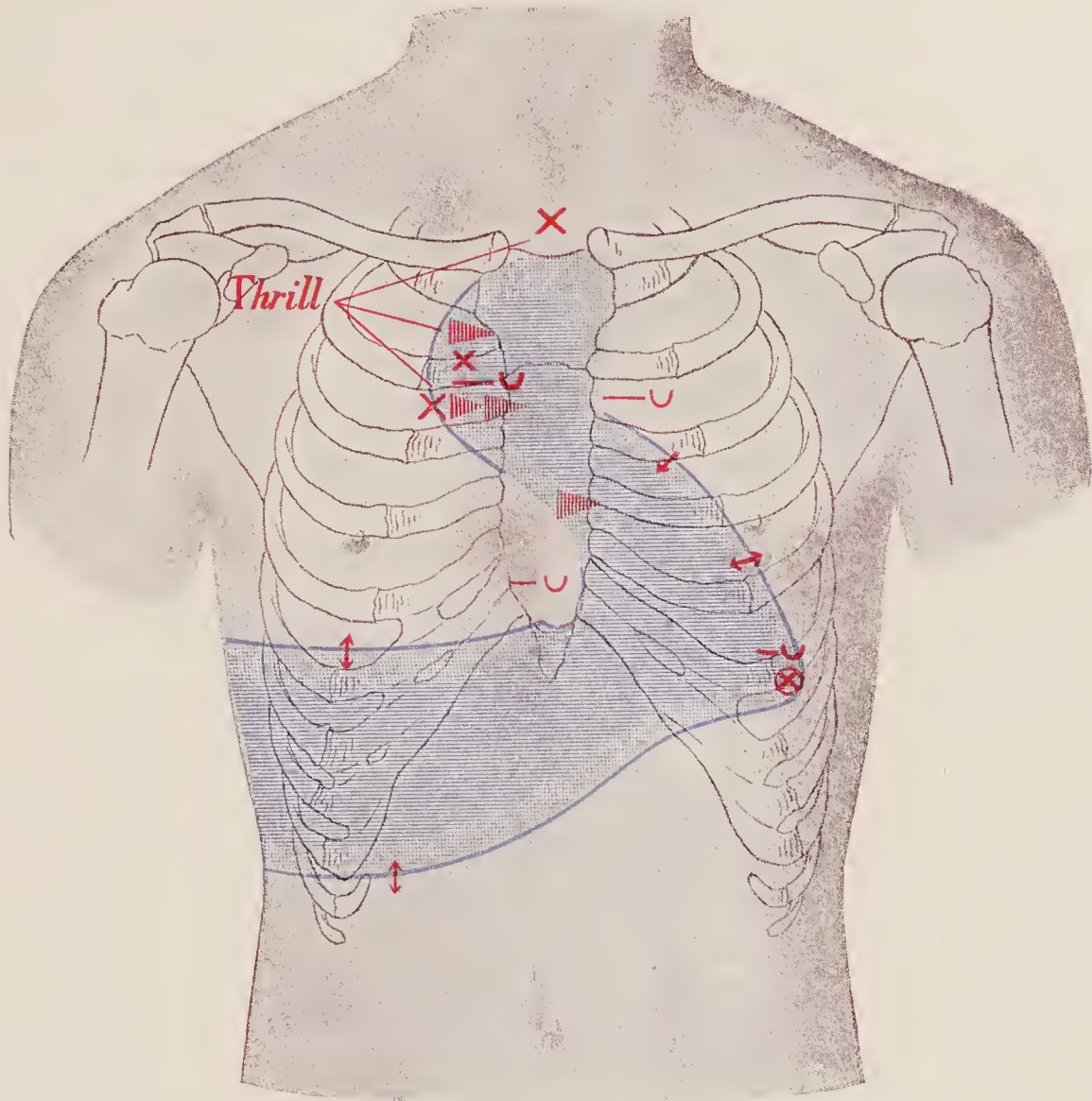


Aneurism of ascending and transverse portions of aorta projecting forward, destroying ribs and sternum. The skin ulcerated, and gradual external leakage took place. The bleeding continued in small amounts for a long time. (Original.)

frequently in aneurism of the transverse portion, due (1) to pressure on the recurrent laryngeal nerves; (2) to compression of the trachea; (3) to compression of the left bronchus. Marked stridor attends the first form. When one of the recurrent laryngeal nerves, more particularly the left, is pressed upon, there is spasm or paralysis of the muscles of the vocal cord, causing hoarseness and loss of voice. Laryngoscopic examination should not be neglected, for paralysis of the abductor muscles without symptoms may be present.

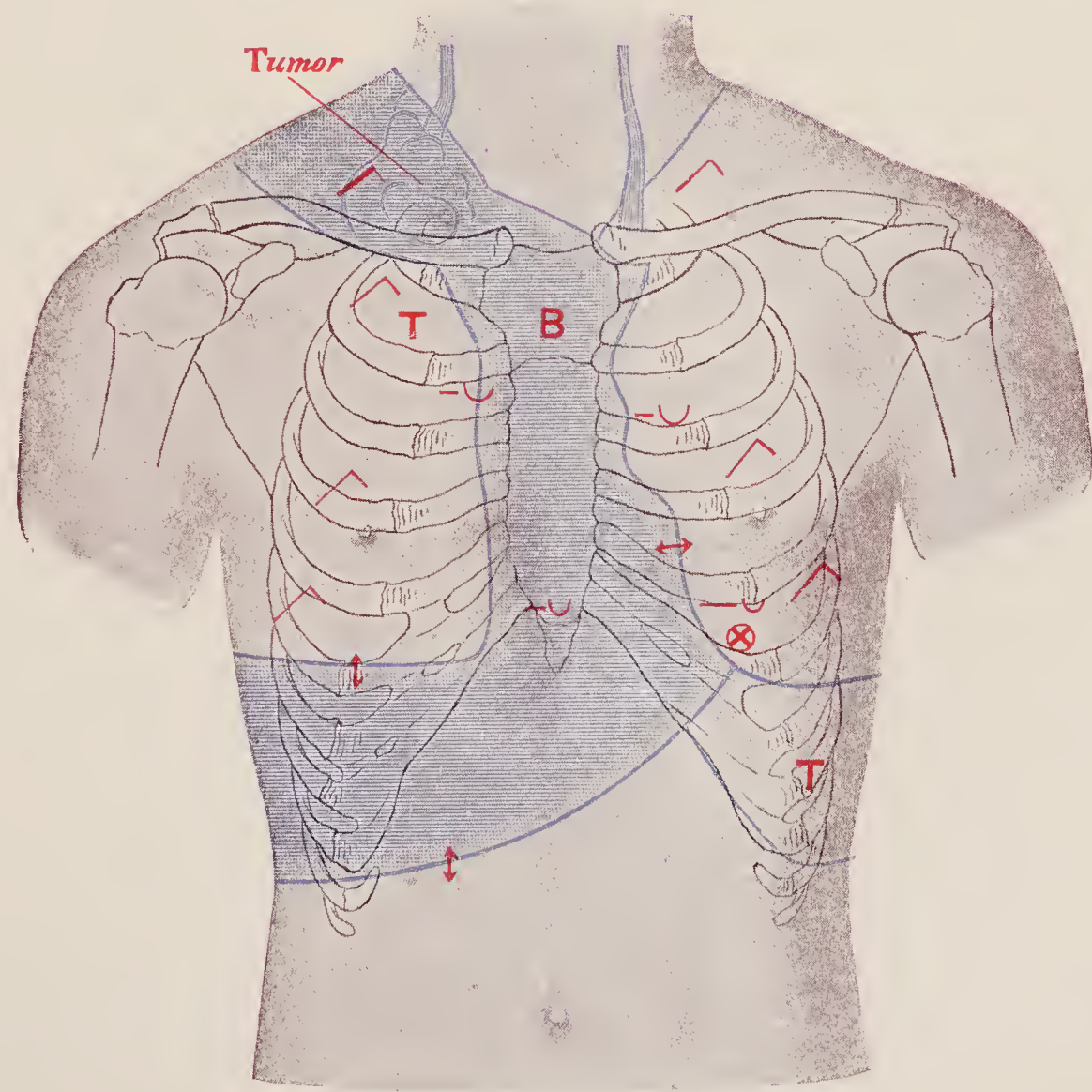
PLATE XXXVII.

FIG. 1.



Aneurism of the Arch of the Aorta.

FIG. 2.

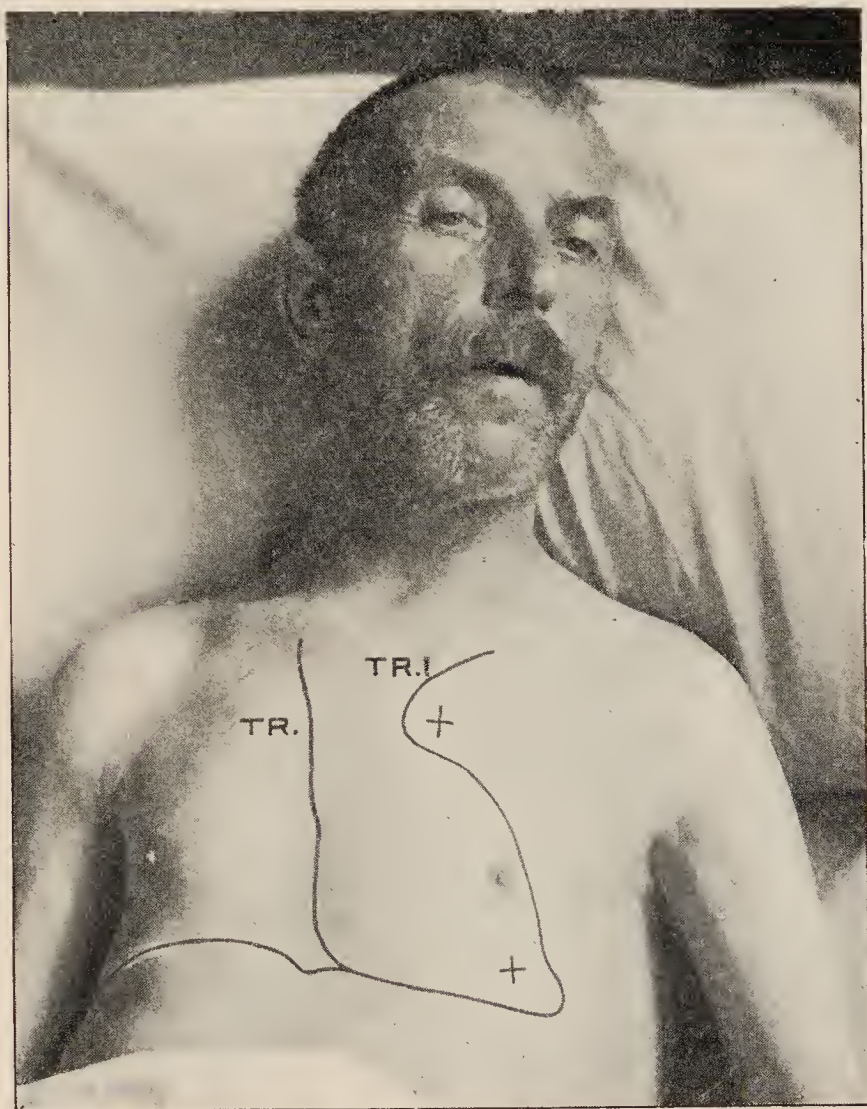


Tumor of the Anterior Mediastinum.

Hemorrhage. The hemorrhage may be gradual when there is slight leakage into the trachea at the point of compression. The amount of blood lost is small. It may take place externally. (See Fig. 352.) Profuse hemorrhages, causing sudden death, occur from rupture into the trachea or bronchus, and from perforation into the lung. With regard to difficulty of deglutition, it may be said that the œsophageal sound should never be passed in suspected cases of aneurism, on account of the danger of rupturing the sac.

Clubbed Fingers. In intrathoracic aneurism clubbing of the fingers and incurvation of the nails of one hand are sometimes, although comparatively rarely, seen.

FIG. 353.



Suspected aneurism. General endarteritis and valvulitis. (Original.)

TR. = Thrill and impulse. + = Murmur. Outline area of dulness. TR'. = In first interspace, thrill and murmur.

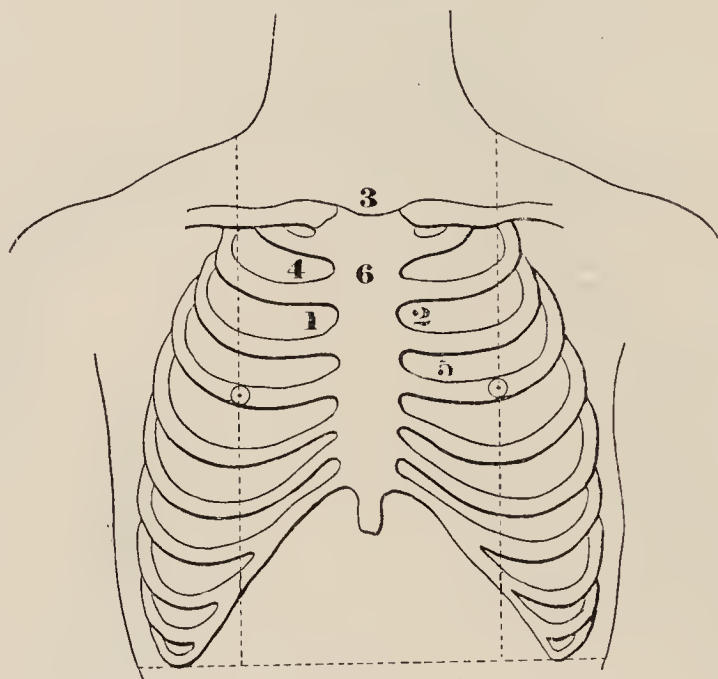
Compression and pressure on the *sympathetic system of nerves* have been referred to. If the pressure is moderate, sufficient only to irritate or stimulate the vasodilator fibres, dilatation of the pupil, usually associated with unilateral pallor of the face, results. If the pressure is sufficient to paralyze or destroy the vasodilator fibres, the pupil becomes contracted, and one side of the face hyperæmic and usually the seat of increased perspiration. Associated with the contraction of the pupil, narrowing of the palpebral fissure and exophthalmos may be observed; the latter occurs rarely, and is attributed in some cases to venous engorgement.

Physical Signs. (Plate XXXVII., Fig. 1.) **INSPECTION.** In health the position of the aorta can not be recognized. Pulsation may be seen

at the episternal notch in rare instances, particularly in women, independently of disease of the aorta; it is due to nervous palpitation. An aneurism may exist without external visible signs. On the other hand, pulsation may be seen at either side of the sternum above the level of the third rib, most commonly in the second interspace on the right side. The impulse may be seen alone without visible swelling; the chest must be viewed from different situations in order to detect it. An oblique light falling on the surface is sometimes necessary. When the innominate artery is involved, the pulsation is observed in the neck, above the sternoclavicular junction or above the sternum.

With the abnormal impulse a swelling or *tumor* is often present. It may be sufficiently large to press the upper portion of the sternum and adjacent ribs forward. In other instances a tumor the size of the half of a lemon may be seen along the edge of the sternum. The most frequent site is the first and second right, or the second left interspace. The skin over the tumor, as in the case of which an illustration is given, may ulcerate and be the seat of persistent small hemorrhages. The *apex-beat* of the heart is displaced downward and outward from pressure.

FIG. 354.



Possible positions of impulse in aneurism, arranged in order of frequency.

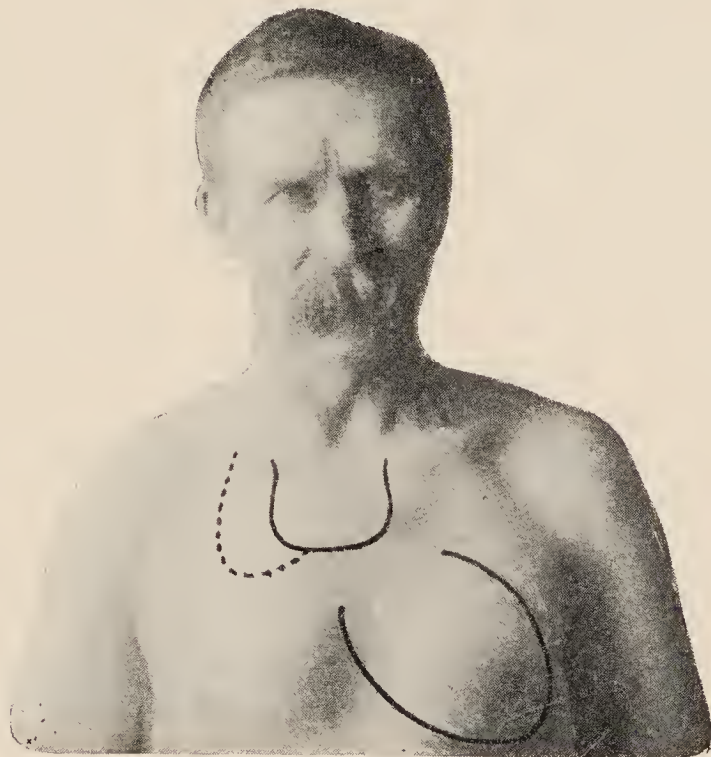
If the aneurism is seated in the ascending portion of the aorta, just beyond the aortic ring, a pulsating tumor may be seen in the third interspace at the right edge of the sternum. If in the ascending portion, beyond the heart, the tumor is in the first or second interspace along the right edge of the sternum. If the aneurism is in the transverse portion of the aorta, the upper portion of the sternum is frequently made to protrude, or the tumor projects upward into the fossa of the neck. If in the descending portion, it is in the second or third interspace on the left side. In this portion of the aorta a tumor is seen in the left scapular region in rare instances.

PALPATION. Palpation must be employed by the usual method; bimanual palpation must also be used, one hand placed upon the sternum and the other upon the vertebræ. Moderate pressure should be exerted.

Palpation should also be employed at different periods of respiration. At times signs are only yielded at the end of complete expiration. It must further be said that palpation must be employed both with the tips of the fingers and with the palm of the hand applied to the surface.

By palpation the area and degree of pulsation are determined. If the aneurism is large or has perforated, the impulse is expansile and heaving in character. The sac may be soft and fluctuating, but usually presents considerable resistance. In addition to the systolic impulse the diastolic shock is also felt. This is a most conclusive physical sign. A thrill is frequently present, systolic in time, usually due to dilatation of the arch; at times, to sacculated aneurism. Without visible tumor, pulsation and thrill may be felt in the suprasternal notch, if the head is bent forward, so that the tissues are relaxed, and the fingers pushed down toward the aorta. When the aneurism is filled or filling with clot, the tumor may be seen and felt, but no impulse will be transmitted to the hand, or thrill be felt by the fingers.

FIG. 355.



Aneurism of aorta.

Area of absolute dulness, dark line. Area of relative dulness, broken line. (Original.)

PERCUSSION. Percussion furnishes the most reliable evidence of the presence of an aneurism or aneurismal dilatation in cases in which the tumor is not too deep-seated or small in size. The dulness may be relative only. (See Cardiac Percussion.) The area of dulness is increased somewhere in the course of the aorta. It may be observed projecting outward at the right edge of the sternum when the ascending portion of the aorta is the seat of disease, or over the entire upper part of the sternum extending toward the left, when the transverse portion is diseased. It may be observed as an extension of cardiac dulness upward in the second and third interspaces. Sometimes dulness is detected in the scapular regions, particularly of the left side. The percussion-tone is flat, and there is marked sense of resistance. Percussion must be employed with the patient in the upright and in the recumbent posture. Eichhorst states that he has made a provisional diagnosis of aneurism in 3 cases, from the

occurrence, during percussion of the anterior chest, of severe paroxysms of coughing, and of the complaint on the part of the patient of severe pain over a localized area. The cough was also induced by palpation of these areas. The necropsy confirmed the diagnosis in each case, and Eichhorst believes that these signs are of considerable diagnostic importance.

Respiratory Percussion. The character of the note and the shape of the dulness must be noted at the end of full inspiration and of full expiration.

Auscultatory percussion is of the utmost value, and the method of percussion taught by Sansom and Ewart must be carefully followed. An aneurismal tumor may be present without thrill or murmur, but yields signs of dulness on percussion.

AUSCULTATION. As just stated, murmurs may not always be present. They depend upon the amount of fibrin in the sac. When present, the murmur is usually systolic in time, heard with maximum intensity usually over the abnormal area of impulse or tumor, or over the increasing area of dulness. It is transmitted in the direction of the vessels, and may be heard louder in the vessels of the neck and along the course of the aorta. Often a double murmur is heard, the diastolic sound being frequently due to associated regurgitation at the aortic orifice. Sometimes the diastolic murmur alone may be heard. Increase in intensity or accentuation of the aortic second sound is pronounced. The sound is ringing in character, and is rarely absent in large aneurisms.

Gerhardt has described a rare arteriodiastolic murmur in the left interscapular region, due to beating of the aneurismal sac against the left bronchus. Glasgow has described a systolic thud or shock in the brachial artery similar to that which occurs in aortic insufficiency.

THE PERIPHERAL VESSELS IN ANEURISM. The pulse in the two radial arteries may show a marked difference both in volume and in time. The difference may indicate the position of the aneurism. If the pulse of the right radial is smaller than the left, the aneurism may be in or near the innominate artery; if the opposite, it is near or includes the orifice of the left subclavian. In the same way the difference in time may also aid in determining the location. Osler refers to obliteration of the pulse in the abdominal aorta and its branches. In one case he could not feel throbbing in the aorta and the femorals, although the circulation was unimpaired. The aneurism was in the descending portion of the aorta, and its pulsation was seen in the left scapular region. The sac was sufficiently large to act as a reservoir which filled during the ventricular systole and emptied in a continuous instead of an intermittent stream, the effect of the ventricular systole being lost.

TRACHEAL TUGGING. Tracheal tugging may be obtained in one of two ways. By the old method the patient should be sitting or standing, while the observer sits or stands to one side, and faces him. With the hand farthest from the patient steadying the head, the observer gently but firmly grasps the surface of the cricoid cartilage with the thumb and finger of the other hand, while the head is slightly thrown back. The head is then flexed, so that the neck is no longer stretched. The patient is then told to hold his breath completely, and any up-and-down move-

ment of the trachea is immediately transmitted to the observer's fingers. One must not mistake the transmitted pulsation in the cervical vessels for such movement; and great care should be exercised to see that the breathing is entirely stopped.

In the other method, as proposed and practised by Ewart,¹ the observer stands behind the patient, steadying the latter's head against his body, and the cricoid is firmly held between the tips of the first or middle fingers. The writer, after considerable experience, prefers this second method, on account of delicacy of touch, firmness of grasp, and comfort to the patient.

Similar to this, which is known as Oliver's sign, is Cardarelli's sign—lateral movement of the larynx. It differs from Oliver's only in the direction of the movement of the larynx and is of similar diagnostic value. In some cases lateral or anteroposterior deviation of the trachea in the neck may be observed, and in other cases the upward and downward movement of the larynx and trachea during deglutition is much restricted. Hall has lately referred to a tracheal diastolic shock which he regards as important in the diagnosis of aneurism.

Diagnosis. The special points of diagnosis are: the ætiological factors; the antecedent pathological conditions, as arterial sclerosis; the occurrence of pain; the occurrence of pressure-symptoms; and the physical signs. These have been sufficiently dwelt upon, and it is not necessary to consider them again. It must not be forgotten that aneurism may be present without diagnostic physical signs, and, on the other hand, the pressure-symptoms may also be in abeyance. If one of the two is present in a male subject past forty, with a previous history of syphilis, gout, alcoholism, or muscular strain, the probability is that an aneurism exists.

Cases are often seen that present physical signs of aneurism which are due to valvulitis and occasionally to pericarditis. The remarkable case from which Fig. 353 was taken simulated aneurism during life, in thrill, impulse, and area of dulness. It was thought that the transverse and upper part of the descending aorta were the seat of an aneurism. Aortic and mitral valvulitis were known to exist. At the autopsy were found chronic endarteritis, large *dilatation of the pulmonary artery* (9 cm. in circumference), aortic and mitral valvulitis, chronic fibrous pericarditis, dilated and hypertrophied left auricle and ventricle, but no hypertrophy or dilatation of the right heart.

The pressure-symptoms always point to some form of intrathoracic disease as the cause of this group of symptoms. Thus in cancerous disease of the lymphatic glands, or other tumors within the mediastinum, pressure-symptoms exactly simulating aneurism may be present and also the physical signs of a tumor. In tumor, however, there is usually absence of certain ætiological factors of aneurism, such as strain, syphilis, etc., and the tumor, if primary, is very common in young subjects. The tumor rarely projects externally, and still more rarely pulsates. If pulsation is present, it is not of the expansile character seen in aneurism, nor is there as decided a systolic shock when the ear is held against the chest. By the same method we observe the shock of the heart-sounds,

¹ British Medical Journal, March 19, 1892.

which are notably lessened or absent in tumors from causes other than aneurism. In deep-seated tumors with pressure symptoms the condition of the arteries, apart from aneurism, is of diagnostic importance. Accentuation of the aortic second sound, with hypertrophy of the heart, points to aneurism. The presence of tracheal tugging is also a valuable diagnostic point in its favor. In tumor, and especially in cancer, there are emaciation and the development of a cachexia, which, as is well known, is most pronounced in cancer of the œsophagus. Most intrathoracic tumors are fatal within a year of the onset of symptoms, while aneurism may last a long time. Cancer of the œsophagus, from its frequent point of election near the left bronchus, often simulates the pressure-symptoms of aneurism.

Schneelee and Porter have described procedures for the recognition of aortic aneurism which they regard as important. A blind rubber bougie is passed down the œsophagus until it is opposite the arch of the aorta or the site of the suspected aneurism. It is then filled with water which may be colored, and a glass tube is attached to the outer end. In health little or no impulse and consequent variation in the height of the fluid are to be noted; in aneurism considerable variations may occur. It has also been held that œsophageal auscultation practised with a solid œsophageal sound results in a murmur being rendered more distinct, and that one ordinarily inaudible may be heard in this manner. It is not difficult to conceive that serious and even fatal results might follow these procedures.

Aneurism must be distinguished from the pulsation of the aorta which is seen in aortic regurgitation. This pulsation is usually associated with dilatation, the latter causing increased dulness, which may add further to the confusion. Exaggerated pulsation without dilatation may, as Bramwell has reported, be the cause of dulness and pulsation over the aorta. The subjects are under forty, neurotic, and usually anæmic.

It is not, as a rule, difficult to distinguish between *pulsating empyema* and aneurism. Wilson points out that aneurism bears a definite relation to the central long axis of the chest. The area of dulness of aneurism is circumscribed, and is usually the seat of murmurs or other sounds synchronous with the rhythm of the heart. The signs of pulsating empyema are usually upon the left side and at a distance from the median line. The percussion-dulness is at the base of the chest and quite extensive. Arterial murmurs are not present. The pulsation is influenced by pressure and by respiratory movements.

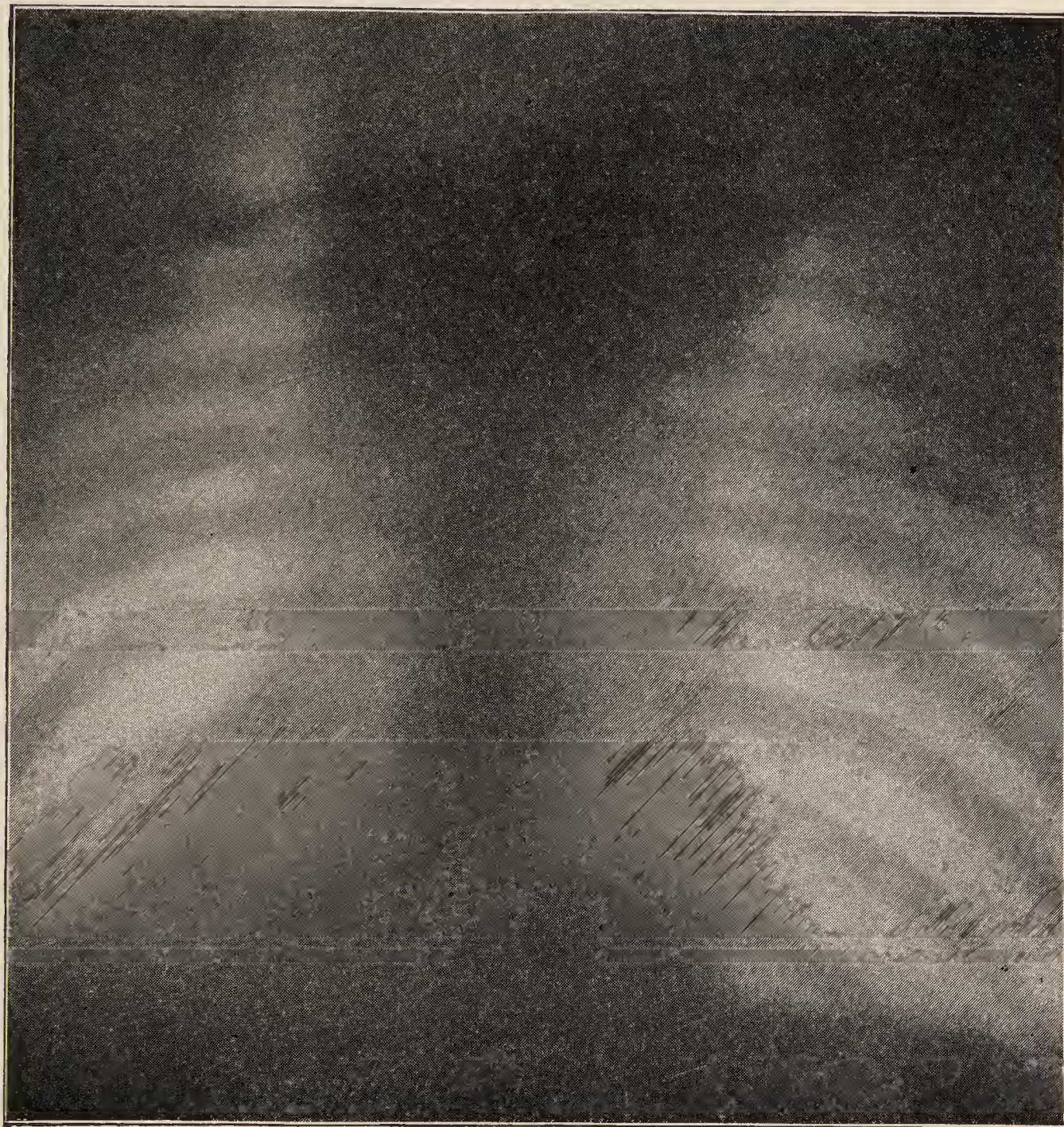
In *mediastinal cancer* we are aided by the discovery of enlargement of the glands in the axilla, neck, or elsewhere, or by a history of the growth in some other area.

Aneurism must not be confounded with *phthisis*. The diseased vessel may occlude a bronchus and cause collapse and bronchial dilatation; hemorrhage may occur; bronchorrhœa and cough always ensue. Fever is not marked, which fact, with tracheal tugging, vascular physical signs, and the absence of tubercle bacilli, points to aneurism.

X-RAY EXAMINATION. By virtue of the large amount of blood and fibrin in an aneurism, the tumor is not pervious to the *x*-rays, and in con-

sequence is readily seen by fluoroscopic examination. Williams and others have been very successful in recognizing an aneurism even when it

FIG. 356



X-ray appearance in aneurism. (PEPPER and LEONARD.)

could not be made out by physical signs. Such examination should be resorted to in all cases. (Fig. 356.)

DISEASES OF THE MEDIASTINUM.

Inflammation of the mediastinum may be limited to the glands or the connective tissue, or may involve both. Moderate inflammation of the glands, lymphadenitis, occurs in bronchitis and pneumonia, particularly if bronchitis is of specific origin, as in measles or influenza. It is said that such inflammation is of common occurrence in whooping-cough, and may be the exciting cause of the paroxysms. DeMussy and Guit  ras have found physical signs of enlargement, characterized by dulness in the upper part of the interscapular region, in cases of this disease and of influenza. Other authorities, as Osler, dispute the possibility of this occur-

rence, or at least of its recognition by physical signs. Tuberculous inflammation of the lymphatic glands of the mediastinum may, however, give rise to local physical signs. Abscess of the glands can not be distinguished during life.

Tumors of the Mediastinum.

Cancer and sarcoma are the most frequent forms of tumor in this locality. Hare found the proportion in 520 cases to be as follows: 134 of cancer, 98 of sarcoma, 21 of lymphoma, 7 of fibroma, 11 of dermoid cyst, 8 of hydatid cyst, and the remainder of lipoma, gumma, and enchondroma. With the application of more correct histological methods we now know that sarcoma is more common than carcinoma. The tumor is most frequently found in the anterior mediastinum when one region alone is affected. The disease may be either primary or secondary. In sarcoma it is usually primary. Males are chiefly affected, and most often between thirty and forty. The tumor may originate in the thymus gland, the lymphatic glands, the pleura, or the œsophagus; the most frequently in the thymus gland.

Symptoms. The symptoms of mediastinal tumor are chiefly due to pressure. *Dyspnœa* is early and constant, and may be laryngeal, or tracheal from pressure on that tube. In some instances encroachment upon the heart or the vessels causes dyspnœa. Again, the dyspnœa may be due to a pleural effusion which accompanies the growths. Cough of a peculiar character occurs. It is laryngeal, and of a dry, brassy quality. Aphonia may arise from pressure upon the recurrent laryngeal nerves. (See Diseases of the Larynx.) If the bloodvessels are pressed upon, symptoms of obstruction occur, depending upon the vessel occluded. Edema of the upper extremities may occur. If the œsophagus is pressed upon, there is difficulty in deglutition. In some instances the sympathetic nerve is pressed upon, causing hyperæmias and pupillary changes.

Physical Signs. The physical signs (Plate XXXVII., Fig. 2) are those of a tumor in the anterior portion of the chest, frequently in the præcordial area, which may or may not pulsate; dislocation of the heart, not limited to any position; great dulness and resistance; frequently conduction of lung-sounds and heart-sounds to some distance; at times a systolic murmur; increased size and pulsation of the veins; and physical signs from pressure. (See Aneurism.) It must be remembered that *pain* is more common in aneurism; *fever* and *emaciation* in mediastinal growths.

Tumors of the anterior mediastinum present, in front, the physical signs of a prominence more or less marked, often including projection of the sternum; an irregular area of dulness; rarely transmitted pulsation; more frequently transmitted heart-sounds and lung-sounds. It is the form in which phenomena from pressure upon the veins are most marked. Symptoms from arterial pressure (difference in pulse), pressure on the vagus and sympathetic are less frequent. *Dyspnœa* may occur.

Tumors of the middle and posterior mediastinum are characterized by

pressure upon the bronchi and structures adjacent thereto ; hence we have symptoms from pressure upon the œsophagus, aorta, and the nerves. *Dyspnœa* and *cough* are the most pronounced symptoms, while phenomena from pressure on the vagus, cardiac palpitation, vomiting, etc., are not uncommon. *Emaciation* and *cachexia* are more marked than in tumors in other regions. Pepper and Stengel consider that fever attends growths in this region with greater frequency. (For differential diagnosis from aneurism, see Aneurism, p. 963.)

Tumors of pleural origin have symptoms of acute or subacute pleuritis, with or without effusion. The fluid secured by puncture is usually bloody, rarely chylous, and may contain suspicious vacuolated epithelial cells. A mass may be suspected if there is great resistance to the trocar. If the tumor ulcerate into the lung, the sputa may contain characteristic groups of cells, while hemorrhagic oozing may be suspicious.

CHAPTER VIII.

DISEASES OF THE MOUTH, INCLUDING THE TONGUE THE FAUCES, PHARYNX AND ŒSOPHAGUS.

THE MOUTH.

THE mouth is affected by comparatively few diseases, and most of these are the result of infection or of trauma, or, rarely, are tropho-neurotic. The cavity is a good breeding-place for all forms of organisms, and were it not for the secretions and constant cleansing of the mouth by the passage of food and its physiological labors, diseases would be very common. Indeed, it is possible that such diseases do not occur at all unless there is such perversion of the normal secretion as destroys its antiseptic or antimicrobial qualities. We know but little specifically concerning the changes in the secretions. Clinically, we do know, however, that in conditions of poor nutrition, in wasting diseases generally, and probably in connection with the rheumatic diathesis, there is such change in the secretions as permits pathogenic micro-organisms to exercise their influence upon the mucous membrane. The result of their action is seen in various forms of *inflammation*.

Diagnosis. All methods of diagnosis are employed. In the *historical diagnosis*, hygienic conditions, food and methods of feeding should be investigated, and the former occurrence of infections are inquired for; these are factors that aid in so much of diagnosis as pertains to ætiology and therapeutics. For the *subjective diagnosis* but few symptoms present themselves, practically only the symptoms of inflammation or of diminished secretion. The *objective diagnosis* is detailed in full in this chapter. By *laboratory diagnosis* we learn the nature of the infections, notably diphtheria, and the condition of the saliva. (See Section VI.) It is readily seen that greater reliance must be placed on objective and laboratory than upon any other method of diagnosis.

Stomatitis.

The *inflammation* is not limited to the mouth, but extends to structures within the mouth, as the gums, and may invade the tongue. It is recognized by the subjective and objective signs common to such inflammations. There is pain, and hence the child (for it usually occurs in children) refuses to nurse or take the bottle, or cries when food is given. The pain is accompanied by *fetor* of the breath. This occurs in all forms of stomatitis.

The usual signs of inflammation are rarely general, being, as a rule, localized to small areas which may rapidly become ulcerated. The mucous membrane is red and hot in the *catarrhal* form; the color

extends to the gums, lips, and tongue. The follicles are enlarged. The tongue becomes red and smooth, or may be covered with a white coating, through which the prominent red fungiform papillæ project. There is increased secretion, which dribbles from the mouth, or is constantly discharged by older patients. The ever red mucous membrane is swollen; the breath is hot and fetid.

A recurring form is seen in gouty subjects. Pain is not marked, but the heat, redness, and burning are severe. Superficial glossitis and salivation are present. The saliva is highly acid, and causes a dermatitis on the chin. Other mucous membranes, as the vaginal, are involved at the same time. An acid mucoid discharge sets up painful irritation at the vaginal outlet.

Aphthous Stomatitis. The areas of intense inflammation are sometimes followed by ulceration. In *aphthous stomatitis* small yellowish-white spots appear, at first discrete, but soon dotted over the mucous membrane inside of the cheeks, in the roof of the mouth, along the sides of the gums, and on the tongue. They subsequently break down into shallow ulcers with raised red margins.

Aphthous ulceration is seen in *foot-and-mouth disease*. It is characterized by greater swelling, with softening and ulceration of the soft parts, than in other forms of stomatitis. There is a history of infection, profuse diarrhoea, followed by constipation, and considerable physical depression.

In young infants that are not well cared for, one not infrequently sees *shallow ulcers*, called "Bednar's aphthæ," on the soft palate or the posterior part of the hard palate. These are due to rough cleansing of the mouth, to the use of bad rubber nipples, or, perhaps, to the pressure of the tongue in nursing and infection of areas thus abraded. They cause no general symptoms, but may interfere with nursing.

Ulcerative Stomatitis. The disease occurs in ill-nourished subjects, and is often intercurrent with exhaustive disease, as chronic diarrhoea. It may be seen in epidemic forms in camps and in penal and other institutions, on account of unsanitary conditions. In ulcerative stomatitis the inflammation is chiefly confined to the gums. They are swollen, red, and covered with ulcers. The gums in which teeth remain are affected, and the ulcers are usually at the gingival border. Gums without teeth are not affected. The ulcers are covered with yellowish material. The flow of saliva is much increased, and it is acid in reaction. The submaxillary glands are enlarged. The fetor of the breath is great.

Parasitic Stomatitis—Thrush. The affection is characterized by raised white patches looking like small curds of milk. The patches vary in size, and on the tongue may cover an area as large as a three-cent piece. (See Thrush.) The white patches are distinguished from milk-curds because they can not be removed with the napkin or brush. The parasite has been called *Oidium albicans* (see Laboratory Diagnosis, Fig. 209); but Forchheimer prefers to group it under the *saccharomyces*.

Stomatitis Materna. Painful ulcers occur in the mucous membrane of the lips and cheeks of nursing women. They are solitary and interfere with mastication.

Gangrenous Stomatitis. The affection appears as a gangrenous in-

flammation of the gums, mucous membrane, and deeper tissues of the cheek. At first a small, dark-red, hard spot is seen, which increases in size and becomes of a purplish color. The cheek rapidly becomes swollen, tense, and brawny. On the surface of the more indurated portions a bleb forms which soon breaks with rapid ulceration. The ulcer is dark and gangrenous and soon perforates the cheek. It extends to the jaw and is followed by necrosis of that bone. The characteristic odor of gangrene attends the process. Gangrenous stomatitis is always secondary to depraved, depressed, or debilitated states of the system. Cases may occur simultaneously in asylums for children in which the hygienic conditions are bad and the food-supply poor.

Mercurial Stomatitis. Mercurial stomatitis, or ptyalism, particularly affects the gums. It also involves the salivary glands. The inflammation is caused by mercury. It may occur from the medicinal use of the drug, particularly in persons who are unduly susceptible, or are not particular in regard to mouth-cleansing. The inflammation is painful and attended by profuse discharge of saliva, hence the name, *salivation*. The tongue is swollen, marked on the sides by the teeth, and may be protruded with difficulty on account of its size. It is tender to the touch, and covered with a heavy, creamy coating. The gums are swollen, red, sore, and bleed on the slightest touch. Ulcers along the border occur, may become diffused, and in some instances extend to the jaw. The teeth become loosened. The fetor of the breath is heavy, offensive, and of a metallic character. The inflammation is usually preceded by a metallic taste in the mouth, and the patient notices pain on mastication, which increases in severity as the inflammation develops. In mild cases it is limited to the gums, in others the tongue and salivary glands and the mucous membrane of the mouth are affected.

Leprosy of the Mouth.

Leprosy frequently invades the mouth. The nodular and ulcerative lesions are seen. It is always associated with the characteristic lesions of the skin. Scrapings or sections would show the characteristic micro-organism.

Glanders.

Glanders may invade the mouth from the nasopharyngeal space.

Actinomycosis.

Actinomycosis results from the entrance of the ray-fungus through carious teeth or an abraded mucous membrane. Often there is first disease of the alveolus, as pyorrhœa, or a periosteal abscess; then the jaw is involved. It may be preceded by a general stomatitis.

Ulcers of the Mouth.

Solitary *ulcers* are seen in *herpes*, secondary to gastric or uterine disturbances, and syphilis. The herpetic ulcers are of frequent occurrence

at the menstrual period or during the course of lactation. The tendency to their formation is often hereditary. I have seen them occur at the menstrual period or in pregnancy in the women of three generations.

In the second stage of syphilis *mucous patches* are seen as bright-red, symmetrical, oval or crescentic patches or erosions, occurring on the mucous membrane, sometimes on the tongue and fauces. They are generally covered with a scanty grayish-white secretion, and are not usually painful.

Sublingual Ulcer. This form occurs on the frænum of the tongue. It is seen in whooping-cough, and is due to the rubbing of the tongue against the teeth in the act of coughing.

Scleroderma.

This rare trophoneurosis occasionally invades the mouth. It is characterized by a submucous infiltration of cartilaginous hardness, the surface of which is denuded of epithelium or covered with crusts. The invasion is through the nostrils or the nasopharynx. Later the infiltration changes to a yellowish-red or a tendinous-like scar.

THE TONGUE.

Inflammation of the Tongue.

Acute glossitis is a rare affection, more common in adults than in children, and more frequent in men than in women. It occurs more frequently in the summer. The onset is rapid. After a short period of tenderness on mastication the movements of the tongue are stiff and painful, or there are pains in the muscles of the neck and submaxillary region. In a few hours the tongue swells; it rapidly enlarges, and at the end of fifteen to twenty hours is three times its natural size, protrudes from the mouth, is indented by the teeth, and is almost immovable, feeling heavy, painful, and tender. It is coated with a thick fur on the dorsum. Salivation accompanies these symptoms, speech is impossible, dysphagia extreme, and dyspnœa not unusual. The glands underneath the jaw are swollen. The temperature rises to 101° F., rarely above it, even if the case is severe.

Death may occur in a few hours from suffocation, or after a longer interval from diffuse suppuration, gangrene, exhausting septic fever, or pneumonia. Gangrene is more frequent than spontaneous resolution. If resolution is to be established, the swelling begins to subside in three or four days. Small ulcers form on the surface of the tongue, and by the end of a week its normal appearance is regained. The fever and distressing symptoms subside with the local swelling. It is said to be due to colds, to bites and stings of animals, to mercury, and to corrosive and acrid substances. It may occur in fevers. The diagnosis is easy. It must be distinguished from the acute œdematous swelling due to salivary calculus or affections of the floor of the mouth.

Acute Ranula. Acute ranula sometimes causes considerable swelling of the tongue, simulating acute glossitis.

Hemiglossitis. Hemiglossitis sometimes occurs. The local symptoms are less in degree. I saw a case in which the inflammation was limited to half the side of the tongue on the posterior surface. It went on to suppuration, but was not attended by serious symptoms, except discomfort in eating. It was preceded by a definite nodule in the substance of the inflamed part.

Glossitis from mercurial poisoning has been described in connection with stomatitis.

Chronic Superficial Inflammation. The surface is smooth and deprived of papillæ over the affected area, which is redder than natural. The margin of the raw patch is sharply defined, but the area has no depth. The epidermis alone is removed. When associated with dyspepsia, it covers a considerable area of the surface of the tongue. The tongue may be deprived of papillæ on the anterior part of the dorsum while the fungiform papillæ remain. It is enlarged and the borders marked by the teeth. The surface looks glossy. The tongue feels stiff and uncomfortable. Movement is irksome, irritating foods are painful; spirits and tobacco cause distress. Indiscretions in diet and slight traumatism quickly produce fresh inflammation. One observer, Hack, has described a form of hereditary glossitis peculiar to women. He observed a row of long, oval areas, caused by previous inflammation. They commenced in early childhood. The tongue was smooth over remaining large areas, with red excoriations here and there. There was no syphilis.

Another form of glossitis is that seen in pernicious anæmia. The papillæ are large, red, and very tender, so that eating, and especially the use of acids, are very painful. The red points contrast with the pallid tongue.

Sequelæ of Glossitis. *Indentations* occur when the tongue is swollen, as in mercurial and other forms of glossitis. The borders of the tongue are indented by the pressure of the teeth; but in states of debility a flabby tongue with indented borders is often seen. Sometimes the swelling is so great that the pressure of the teeth causes ulceration.

Furrows, or grooves and wrinkles, are seen on the dorsal aspect of the tongue. They are not necessarily tokens of disease; in many healthy persons they are of constant occurrence. Furrows vary from a few lines to an inch or more in length. They may be longitudinal, transverse, or irregular. The median furrow is liable to become ulcerated on slight provocation. The edges of the fissures are smooth and without papillæ or fur. Other furrows are directed horizontally and vary in depth. They may be curved and forked. They are more frequent in older persons, especially if the tongue is too large to lie within the circle of the teeth. They are an evidence of past inflammation, or rarely of hypertrophy. They resemble the median furrows as regards smoothness and absence of fur.

Inflammatory furrows occur in chronic superficial inflammation, but more commonly after chronic inflammation with enlargement. The furrows are sometimes so abundant that the surface of the tongue looks like the eyelid. The raised areas become sore, irritated by a foreign body (food) or a tooth. They are an indirect result of inflammation.

True inflammatory furrows, described by Wunderlich, occur. *Dissecting glossitis* is only a more aggravated form of superficial glossitis, or may be due to syphilis. *Fissures* and *clefts* are frequently caused by the rubbing and deep indentation of a rough and jagged tooth. The area around the fissure is inflamed, its base indurated, and the sides and bottom ulcerated. It is recognized by its relation with the offending tooth. It may be mistaken for syphilis.

Syphilis of the Tongue.

Fissures are found on the borders of the tongue; in *secondary syphilis* they are almost certain to occur if the teeth irritate the border. They may be due to the ulceration of a mucous tubercle which is developed upon the border of the tongue. The ulcer is stellate, and gradually deepens until it becomes a foul fissure. Two processes cause the ulceration—the specific infection and the irritation of the teeth.

Syphilitic *ulcers* are not so angry as non-syphilitic sores and fissures, which may occur in poor health. They may be sensitive, however, on account of their location on the tongue. The absence of active inflammation, the large number of sores and fissures, and the association with other lesions of the disease upon the tongue, cheeks, and lips, point to their syphilitic origin. Tertiary syphilitic ulcers are more pronounced and deeper than other forms. They may be as long as two or three inches; they are sinuous and branched.

Gummata may occur on the tongue at the same time. The gummata may be circumscribed or linear, and may break down and ulcerate.

Sclerosis of the tongue, as described by Fournier, follows the healing of these ulcers. It is curious to note that the lymphatic glands are seldom enlarged in association with syphilitic fissures.

Diagnosis. The fissures must be distinguished from *carcinoma* and *tuberculosis*. In carcinoma there is a distinct tumor, which may become fissured. Tuberculous ulceration is a sign of the presence of tubercle in other organs. The tuberculous fissures are small, at first single; tubercle, however, rarely begins as a fissure, but as an ulcer on the tip or borders of the tongue. The ulcers are stellate or irregularly branched; they are shallow at first, and deepen later, but do not widen in a corresponding manner. The lymphatic glands are always involved. (See Tuberculous Ulcer.)

Atrophy of the Tongue.

Atrophy of the tongue is very unusual. Hemiatrophy may occur as the effect of central or peripheral causes, as softening, hemorrhage, or tumors in the region of the hypoglossal nucleus. Other centres near the nucleus are affected, hence other forms of paralysis are seen, due to the lesions of the medulla. These are seen in progressive muscular atrophy and bulbar paralysis, and in hemiplegia. It is not difficult to recognize atrophy on inspection. The functions of the tongue are not affected.

Hypertrophy of the Tongue.

Enlargement of the tongue, or *macroglossia*, is generally congenital, but may occur late in life. The tongue enlarges, and presents pressure-symptoms due to such enlargement. Hypertrophy of the tongue is sometimes seen in idiots and cretins. The hypertrophy is more frequently the result of lymphatic obstruction, on account of which there is lymph-stasis. The diagnosis is easy. Inflammatory hypertrophy occurs in stomatitis, and syphilitic hypertrophy occurs with gummata.

Angina Ludovici.

Angina Ludovici is characterized by slight inflammatory congestion of the throat out of proportion to the symptoms of the inflammation in the external structures. Wooden induration of the connective tissue, which will not pit on pressure; spreading of this induration, which is circumscribed, so that it is bound sharply by unaffected cellular tissue, are characteristic. The induration may extend from the rami of the jaws to the cheek. There is a hard swelling in the *tongue* and along the lower jaw, causing thickening of the floor of the mouth. This is observed by palpating with the finger in the mouth. The glands are not affected. For a long time the nature of this affection was not known. It is now believed to be due to actinomyces.¹

THE FAUCES AND PHARYNX.

The passage-way between the mouth and the respiratory passages is lined with mucous membrane, which is subject to the same diseases as mucous membranes in general. The symptoms thereof are similar to the symptoms of mucous membrane inflammation elsewhere. The large muscles of the pharynx which aid in deglutition are subject to affections which belong to muscular tissue generally, hence *rheumatic inflammation* and loss of power of muscle, or *paralysis*, occurs.

Paralysis of the pharynx has not the same practical importance in the diagnosis of central lesions as paralysis of other structures, such as parts of the larynx. This is due to the fact that the nerve-supply of the pharynx is derived from a nerve (glossopharyngeal) which supplies other structures, paralysis of which is more evident than pharyngeal paralysis, more readily ascertained, and which causes more pronounced symptoms. (See Cranial Nerves.) From its exposed situation the pharynx is particularly liable to *infection* from micro-organisms. The infection may extend from the mouth, or from the nares above, or the micro-organisms may affect it primarily.

The fauces and pharynx may be the seat of morbid processes which occur secondarily to diseases in other portions of the body with a moderate degree of frequency. Inflammations of the mucous membrane of the pharynx are of rheumatic or gouty origin in a large number of cases.

¹ See Parker, *Lancet*, 1879; and Anderson, *Transactions of the Medico-Chirurgical Society*, 1891.

Indeed, gouty inflammation of the pharynx seems to be more common than gouty inflammations of the mucous membranes in other situations. The large majority of subacute or chronic pharyngeal inflammations are secondary to dyspepsia. They also occur from extension of the disease from cavities related to the pharynx.

Affections of the tonsils are usually more common in rheumatic states, and bear some relationship to the rheumatic diathesis. Inflammation of the tonsils may follow acute rheumatism or may alternate with it. A patient who is predisposed to rheumatism may at one season have tonsillar inflammation, at another rheumatism. The writer has seen tonsillitis immediately followed by rheumatism, and then the latter replaced by the former.

The not infrequent occurrence of endocarditis with tonsillitis has been used as evidence of the rheumatic nature of the tonsillar disease, and it is very probable that the infection which causes rheumatism frequently enters through the tonsils. Packard considers that the tonsils may reduce the virulence of infections entering through them, and that this attenuation results in the production of rheumatic symptoms rather than of ordinary septicæmia.

There is also good evidence that tuberculosis sometimes has its primary seat in the tonsils, and may be disseminated from them, when it is especially likely to involve the cervical glands secondarily. In these primary cases it is a food infection sometimes. Secondary tuberculosis of the tonsils is apparently not uncommon.

Apart from what has just been said, diseases of the pharynx bear little, if any, diagnostic relationship to disease elsewhere. While there may be cyanosis of the mucous membrane, or tuberculous ulceration, or other changes which we have noted, the signs of the primary disease are so much more marked that we need not rely upon the appearance of the pharynx or symptoms of pharyngeal disease for diagnostic purposes. The only general affection which may be diagnosticated from the appearance of the pharynx alone is measles. In obscure cases of sudden fever, with nasal catarrh, the appearance of the eruption in the situation just indicated may lead to the recognition of measles when the external eruption is not apparent. For the purposes of the therapist it should be borne in mind that symptoms referable to the pharynx are frequently due to disease in the nares, particularly in that portion of the pharynx which is not open to direct inspection—the nasopharynx.

The *general symptoms* of pharyngeal disease are not marked, except in diphtheria, in erysipelas, in retropharyngeal abscess, and in affections of the tonsils. In the latter the general symptoms appear to be out of proportion to the local process. The high fever, the intense headache and backache, and rapid pulse, seem to point to a process which in extent and severity should far surpass that which occurs in the tonsils.

As the structure is a passage-way or channel, affections of the pharynx are liable to obstruct it, causing symptoms of *occlusion*. As it is a channel for the passage of air, obstruction in the pharynx will lead to *dyspnœa*. (See Chapter IX.) In addition to its function as a simple channel, the pharynx is concerned in deglutition. When, therefore, there is obstruction

of the pharynx, *deglutition* is made difficult, or may even become impossible.

Attention can not be too strongly directed to the investigation of the *nasopharynx* in children who are poorly developed physically and mentally. This combination of symptoms is often due to adenoid vegetations of the nasopharynx.

Diagnosis. The diagnosis of diseases of the pharynx is based upon the facts gleaned by all methods of diagnosis.

Historical diagnosis confirms other methods in the diagnosis of pharyngitis and tonsillitis (rheumatism), and the infections.

In the *subjective diagnosis* we consider cough (Chapter X.), pain (Chapter VIII.), dyspnœa (Chapter IX.), and dysphagia (Chapter XIII.).

The *objective* and *physical* diagnosis are considered in full, for by these methods and a consideration of the clinical course of the disease the diagnosis is most completely established.

By *laboratory diagnosis* we establish the presence or absence of infections, as diphtheria, tuberculosis, and leptothrix.

THE TONSILS.

Acute Tonsillitis.

Acute inflammation of the tonsils may affect the follicles, to which form the term *follicular tonsillitis* is applied, or it may be limited to the mucous membrane, when it is known as *catarrhal* or *erythematous tonsillitis*. If with the catarrhal inflammations vesicles appear on the surface of the mucous membrane, the term *herpetic tonsillitis* is used. When the inflammation extends to the stroma of the glands it goes on to suppuration. It is characteristic of all forms of acute tonsillitis to recur frequently in the same subject. The relationship to rheumatism, which applies to both the acute and the suppurative forms, has been spoken of. The various forms of tonsillitis occur at any period of life, although it is least common under ten years of age; the suppurative form occurs most frequently in adolescence. Tonsillitis occurs in both sexes. It may follow exposure to wet and cold, although patients who are subject to the attacks bear exposure well unless they are at the same time unduly fatigued. The *follicular* form of tonsillitis is apparently associated with faulty drainage or other unhygienic conditions, and hence it is possible that noxious emanations act as an exciting cause. Several persons of the same family may be affected at one time, so that it is often difficult to distinguish the cases from diphtheria. The disease, however, is usually not contagious. Persons brought in contact with the family, but who do not reside in the same house, escape the disease; this applies as well to children, who would, if the cases were diphtheritic, be most liable to become infected; actual epidemics of tonsillitis, due usually to the streptococcus, occur in rare instances, however, and in such cases the diagnosis must rest upon bacteriological examination. The disease occurs more commonly in the spring than in any other season of the year, more especially in cold and wet seasons.

Symptoms.—In *follicular tonsillitis*, with or without a rigor, but always

with chilly sensations, the temperature rises rapidly to a great height. The subjective sensation of fever is quickly noticeable to the patient, and is generally more pronounced than in other affections. With the chill and during the rise of temperature there are some frontal headache and severe pain in the back and limbs. The pain in the back is most excruciating. In a short time the patient complains of pain in the throat. Swallowing is difficult, and there is a sense of fulness. The throat is dry and burning. On examination the tonsils are found to be swollen, and a yellowish-white exudate is seen in the crypts. In twenty-four hours the points may coalesce to form a patch. The glands expand slightly, and may extend only slightly beyond the arches, or, in younger subjects, one-quarter of the way into the lumen of the fauces. Sometimes one gland is affected before the other. The difficulty in deglutition increases and the voice becomes nasal. There is usually some enlargement of the cervical glands. The general symptoms continue for forty-eight hours, the temperature remains at 105° F., and the pulse is very rapid. After the first twenty-four hours the pain in the back lessens. The tongue is coated and the breath heavy. The urine is loaded with urates. At the end of the fifth day the fever, which subsides gradually, has disappeared. The local symptoms, however, may remain longer—that is, the tonsils are still enlarged and the exudate disappears slowly. Sometimes the prostration and general symptoms are so severe that after the fever has subsided convalescence may be protracted.

Albuminuria, due in all probability to the fever, frequently occurs; in some cases, undoubtedly, acute nephritis attends the attack and retards the convalescence. In a case under the writer's care the patient first had acute rheumatism; this was replaced by a severe attack of tonsillitis, during which albumin, blood, and granular casts were found in the urine. The swelling of the tonsils subsided in due time, but the nephritis continued for a long period, finally ending, however, in complete recovery.

In *herpetic tonsillitis* the severe pain and intense general symptoms are out of proportion to the local lesion.

In *suppurative tonsillitis* the constitutional disturbance is also very great. The temperature rises high, 104° to 105° F., and the pulse is very rapid, varying from 110 to 130 in the adult. The inflammation usually begins in one tonsil, and the other may be involved later. The tonsils at first are enlarged and firm and red. There is swelling of the surrounding tissues. In twenty-four hours deglutition becomes almost impossible, and there is salivation. At the end of forty-eight hours the patient presents a striking appearance. The glands of the neck are enlarged, the patient is unable to open his mouth, the voice is nasal or almost suppressed; there is dribbling of saliva from the mouth. The face may have a dusky hue in spite of the capillary congestion due to the fever. There is constant desire to discharge saliva and accumulated secretions from the back part of the mouth. The patient can not lie down. The pain is extreme, and is aggravated by swallowing. It is sometimes of a throbbing character, and often shoots to the ears—indeed, earache may be the chief complaint. The patient does not take food,

and exhaustion soon ensues. During the twenty-four hours before rupture takes place the previously reddened face becomes blanched from exhaustion. The fever is continuous during this time, with great rapidity of the pulse. The patient may be delirious. Sometimes the delirium is marked and the patient resists efforts to keep him in bed.

The suffering is out of proportion to the severity of the case. About the fourth or fifth day suppuration ceases, and if the finger can be inserted into the mouth between the almost closed teeth, fluctuation is detected. In cases in which the mouth is opened a little more freely, in addition to the swelling of the tonsils below the arches, marked swelling and projection forward of the half-arches may be seen. The fluctuation may be detected through the anterior fold of the palate, and, if lancing is to be performed, the pus can only be reached through this structure. In short, a peritonsillitis takes place. After spontaneous rupture, which usually occurs into the mouth, instant relief is experienced. Rupture may take place into the pharynx and cause suffocation from entrance of pus into the larynx. In rare cases it has opened into the carotid artery, causing instant death from hemorrhage.

Diagnosis. The diagnostic features of acute tonsillitis are the sudden high fever, severe backache and headache, pain in the throat, and albuminuria. The characteristic appearance of the face, the salivation and pain, with suppressed voice and difficult deglutition, should distinguish it from trismus or *tetanus*. In both, the jaws are closed. It must not be confounded with *smallpox*, which it resembles during the first twenty-four hours.

Cases of follicular tonsillitis are frequently mistaken for *diphtheria*. The follicular inflammation in tonsillitis is limited to the gland, on which patches of a yellowish-gray color, easily removed without leaving bleeding surfaces, are seen. In diphtheria the membrane is of an ashy-gray color, not in points or small patches, or separated by red tonsillar tissue; it extends to the pillars of the fauces, and may appear on the uvula. There are, nevertheless, many cases which are doubtful, and in which a bacteriological diagnosis must be resorted to. (See Bacteriological Examination.) A history of exposure sometimes helps to arrive at a conclusion. The cases that particularly increase our anxiety are those of adults who are subject to attacks of follicular tonsillitis. In the grave and extensive forms of diphtheria with asthenic symptoms (septicæmia) the diagnosis is not difficult.

Enlargement of the Tonsils—Chronic Tonsillitis.

The tonsils may be enlarged on account of repeated attacks of acute inflammation or from chronic inflammation. They do not appear to cause serious symptoms unless associated with adenoid vegetations in the nasopharynx. They may interfere with hearing, however, and with breathing, and cause snoring at night. Fetor of the breath may be noted, particularly if the secretion lodges in the crypts. The latter may be recognized by its characteristic yellowish color and by its odor on removal. The enlarged tonsils are irregular in contour.

Foreign Bodies in the Tonsils.

Foreign bodies in the tonsils are not of common occurrence. They give rise to local symptoms, as the sensation of the presence of a mass causing repeated efforts at swallowing. If calculi are present, the patient may complain of a rough sensation. Calculi follow frequent attacks of quinsy. *Hydatids* are sometimes located in the tonsils.

Adenoid Vegetations of the Nasopharynx.

Adenoid vegetations cause more or less obstruction in the nasopharynx. The symptoms may be classed as primary and secondary. The former are local, and due to the foreign substance *per se*; the latter are local and general. The former are catarrhal; the latter the result of stenosis.

Local Symptoms. In a large number of cases there is *discharge* from the nose. This may be mucopurulent or be associated with crusts. If the discharge is not constant, the child is subject to coryza, with its customary discharge, on the slightest provocation. Even without the chronic purulent nasal discharge, mucus and blood may be passed at night and be found on the pillow in the morning.

The *hearing* is frequently impaired. There may be simply dulness of hearing, or it may amount to marked deafness, either because of pressure of the adenoid vegetations or extension of secondary inflammation to the Eustachian tubes. The senses of *taste* and *smell* are often much impaired. There is increase in the secretion of pharyngeal mucus, which in older persons causes difficult expectoration.

Rhinoscopic Examination. The roof of the pharynx is covered with rounded or villous projections, often concealing the posterior nares. Rarely the villi may be seen projecting below the soft palate. In children the examination is difficult, and hence digital exploration must be used under an anæsthetic. The finger readily detects the masses, which sometimes are soft, at other times tough and of fibrous or cartilaginous consistency.

The Appearance. The *expression of the face* is characteristic. It is dull and stupid, and may be drawn. (Figs. 357, 358, 359.) The *mouth*

FIG. 357.



Appearance in adenoid disease.
(DAWSON-WILLIAMS.)

is kept open in breathing. The lips are thickened, dry, and may be cracked. The palatal arch is high and narrowed.

The *nostrils* are flattened laterally. Rarely they may be depressed. In one instance, which the writer saw with Harrison Allen, the exterior of the nose suggested inherited syphilis, especially because of our knowledge of the possible presence of the disease. There were no other evidences of hereditary syphilis in the child or in any member of the family.

The *voice* is thick and muffled, becoming indistinct upon the occurrence of slight cold.

The Chest. While there is a general lack of physical development, the appearance of the chest is most striking; the cases have been fre-

FIG. 358.



FIG. 359.



Types of the "adenoid" facies. (POSEY and WRIGHT.)

quently mistaken for rickets. In this country adenoid vegetations are a common cause of chest deformity, whereas in England and on the continent rickets is the most frequent cause. The ribs are prominent in front, the sternum is angulated forward at the manubrio-gladiolar junction and grooved at the gladiolar-xiphoid junction. A saucer-shaped depression is found at the lower costal cartilages. The ribs behind are closely compressed, so that the intercostal spaces at the lower part of the chest are obliterated. The chicken-breast appearance is most striking, with the depression in the lower portions of the chest. During inspiration the diaphragm may be drawn inward in the middle and lateral thoracic region.

In addition to the "chicken-" or "pigeon-breast" the more advanced deformity known as the "funnel-breast" or *Trichterbrust* is seen. In children who suffer from asthma and bronchitis the chest becomes emphysematous.

Mental and Nervous Symptoms. Headache, listlessness, and indisposition for mental exertion are marked. The patients are usually backward in their studies and are unable to fix their attention for any length

of time upon any subject. The child is forgetful and can not study without effort. *Aprosexia* is the term applied to this condition.

Choreiform *spasm* of the face occurs in connection with it. *Enuresis* is a frequent associate symptom. The child is subject to frequent attacks of *indigestion*. I have seen the following occur in many cases: Prior to operation the child had an abnormally poor appetite and was subject to frequent attacks of indigestion, characterized by vomiting, with fever. After the operation the appetite improved and continued good, and the indigestion ceased entirely. The cases had been under observation for a number of years before and after the operation. The indigestion seems to have been due to the fact that, owing to the obstruction, the child would have to eat rapidly in order to keep the lumen of the mouth free for breathing purposes. The rapid eating, of course, prevented proper mouth digestion, and hence the occurrence of gastric catarrh.

Symptoms from Embarrassed Respiration. In addition to mouth-breathing, the patient snores at night, and sleep is always disturbed. The respirations are irregular, with a pause between, followed by noisy inspirations. The difficulty of breathing is the cause of restlessness, and the child will often wake up in the night with dyspnoea. *Night-restlessness*, with *dyspnoea* and irregular respirations should therefore suggest obstruction in the nasopharynx.

Diagnosis. Diagnosis is based upon the facies, which is very characteristic, and the physical examination. In children digital examination is necessary. The finger can readily detect small flat bodies or grape-like masses in the nasopharynx.

The student can not become too familiar with the symptoms and signs of adenoid disease of the nasopharynx. There is no doubt that in large cities this affection is of more common occurrence and more disastrous in its results than any other that we have to deal with in children. It may be said that in children in poor health, anæmic, with impaired digestion, and lack of muscular and physical development, if the causes are not due to impure air and improper diet, or to improper sanitation generally, it is almost certain that there is disease of the nasopharynx. The writer has seen a large number of cases in recent years in his practice, and has had the satisfaction of seeing the entire picture of the child change after proper operations. It may be said in passing that this change does not take place at once, but after three to twelve months the child will be fully restored in physique, if during that time attention is paid to proper exercise and the development of the chest. Notwithstanding all this, however, the natural shape of the chest and appearance of the face are only resumed gradually.

Inflammations of the Pharynx.

Acute Pharyngitis. Inflammation of the pharynx, or sore throat, follows cold or exposure, particularly after the patients have been physically depressed. The acute inflammation may be associated with rheumatism or gout, and often involves the tonsils as well as the pharynx. The symptoms are pain on swallowing, with dryness and a constant

desire to hawk and cough, on account of the tickling sensation. There may be slight laryngitis and inflammation of the Eustachian tubes, with deafness. Stiffness of the neck and enlargement of the cervical glands attend the local inflammation. The general symptoms are not marked. The attack is ushered in by chilliness and slight fever. On examination the mucous membrane is seen to be congested, dry, and glistening, and covered in spots with sticky secretions. The uvula may be swollen considerably. When the submucous tissues are involved, the parts are more swollen and there is great dyspnœa. The dysphagia is more marked, although the pain is not any greater. The fever is higher. The larynx is always involved, causing aphonia.

Phlegmonous Inflammation. A diffused inflammation of this character occurs. The writer saw one case with dyspnœa, nervous symptoms, and high temperature, simulating severe pneumonia. Pneumonia was thought to be present because there were congestion and œdema of the lungs. This occurred during the prevalence of a recent epidemic of influenza. The disease began in the pharynx; the tissues were swollen and infiltrated. The early symptoms were pharyngeal. The dysphagia was extreme, and there was an abundant mucopurulent expectoration which did not contain pneumococci. Death took place on the ninth day from exhaustion. The autopsy showed a high degree of congestion of the lungs, and phlegmonous inflammation of the pharynx, larynx, and trachea. While, therefore, the recognition of an acute phlegmonous inflammation is not difficult, it must not be forgotten that it is a grave disease, which may present such marked pulmonary and systemic symptoms as to lead to the suspicion of pneumonia.

Angina Ludovici. Angina Ludovici is an inflammation of the cellular tissue of the floor of the mouth and neck. It is probably a form of actinomycosis. The swelling is most marked below the jaw of one side. The symptoms are intense and both local and general. There are general septic symptoms from the outset. With the swelling there are œdema and board-like induration. Redness and the rapid formation of an abscess occur rarely. The throat is not affected. Death takes place from reflex suffocation or in coma. (See the Mouth.)

Rheumatic Pharyngitis. Rheumatic pharyngitis is of short duration, without unusual objective symptoms. Pain is intense, deglutition difficult. The usual concomitants of rheumatism are present. It frequently gives place to torticollis, lumbago, or rheumatism in some other situation.

Chronic Pharyngitis. Chronic pharyngitis follows acute attacks, and is a frequent accompaniment of nasal catarrh. It is common in smokers and alcoholic subjects; the use of the voice in loud tones, as by clergymen, auctioneers, etc., is also a cause. It is a frequent attendant upon indigestion, due probably to the eructations. The objective signs are relaxation of the mucous membrane, with dilatation of the veins. The membrane is covered with a thick secretion, which is dry and glistening. In the granular form the wall of the pharynx is covered with millet-seed projections and is congested. Tough mucus is seen in small areas.

Retropharyngeal Abscess. The inflammation may begin in the submucous connective tissue, and a retropharyngeal abscess forms. There are

high fever and dysphagia, with stiffness of the neck and enlarged glands. On examination a projection into the pharynx can be seen or distinctly felt on the posterior wall. The disease may be difficult of recognition in infants, in whom it is not possible to get a good view of the pharynx. On the other hand, it may be simulated by disease of the cervical vertebræ, in which there may be stiffness, difficulty in deglutition, and possibly a tumor. It must not be forgotten that retropharyngeal abscess may result from caries of the cervical vertebræ. In children the abscess is attended with dyspnœa and alteration in the voice, so that laryngeal disease may be suspected. I recall a case of retropharyngeal abscess in which the dyspnœa was so severe as to suggest croup; in fact, preparations for tracheotomy were made, when sudden rupture of the abscess revealed the nature of the disease. Fortunately the child had been kept in the upright position, so that pus was discharged into the mouth, or suffocation would have ensued.

INFLAMMATION OF THE PAROTID GLAND.

First, specific inflammation or parotitis (see Mumps); second, symptomatic parotitis occurs in typhoid fever, pneumonia, pyæmia, and septicæmia. The process is intense, characterized by swelling, redness, and heat over the parotid gland. There are pain and difficulty of mastication; suppuration rapidly ensues in the septic form. It is thought to be an unfavorable symptom, but I have seen two cases in typhoid fever get well. In a case of septicæmia it did not advance to suppuration. Stephen Paget has described a symptomatic inflammation in disease of the abdomen and pelvis. He collected 101 cases, 50 of which were due to injury, disease, or temporary derangement of the genital organs, as by slight blows, or in females to the introduction of a pessary. It may occur before the menstrual period or during pregnancy. Septicæmia or pyæmia does not attend the process—indeed, many of the cases are afebrile. In 78 cases, 45 suppurated and 33 resolved without suppuration.

Gowers describes a case of parotitis which occurred in the course of fatal peripheral neuritis.

THE ŒSOPHAGUS.

Inflammations of the Œsophagus.

Acute Inflammation. Acute inflammation is recognized by severe pain on swallowing. It is associated with the sensation of a foreign body in the lower portion of the throat. There may be tenderness on pressure along the course of the Œsophagus. The pain is aggravated by speaking. The pain may extend along the vertebral column to the cardiac end of the stomach, and is usually of a burning or raw character. When the inflammation is due to traumatism, as the swallowing of acids or other caustics, the mouth and pharynx show the effects of the inflammation, and, in addition, there is agonizing burning pain at the root of

the neck and between the shoulders. The inflammation is usually attended by erosion of the mucous membrane, and hence not only frothy mucus of a glairy character is expectorated, but also blood and shreds of membrane. The effect of the corrosive poisoning on the general system is marked. There is great prostration. Because of the accompanying gastritis there is intense thirst. Acute inflammation of the œsophagus may end in ulceration or in resolution. The traumatic inflammation is followed by chronic inflammation, which ultimately results in stricture.

Chronic Inflammation. Chronic inflammation is attended by pain in the act of swallowing; liquids are swallowed readily, but solids with great difficulty. Viscid mucus is expectorated, usually in large amounts.

Abscess of the Œsophagus.

The acute inflammation may terminate in abscess. The abscess usually develops slowly, with pain on swallowing and on movements of the neck. When the abscess is high up in the gullet, it may present on the exterior of the neck. If it is situated outside of the œsophagus, and is secondary to disease of the vertebræ, it is slow and chronic in its course; fever and rigors attend its development.

Ulcer of the Œsophagus.

Ulcer, excepting in connection with acute inflammation, occurs but rarely. The so-called "decubital" ulcer is most common. This forms in the later stages of life in old people or in those who are greatly reduced by long illness, and the symptoms are usually marked. Its site is opposite the cricoid cartilage, the position where the œsophagus is most subject to injury. In rare cases typhoidal ulceration occurs, and may give rise to ultimate stricture. Somewhat more commonly a *peptic* ulcer may form in the lower part of the œsophagus. The symptoms with these forms are chiefly localized pain and pain on swallowing, occasionally complicated by hemorrhage, or rarely by perforation. They may lead to stricture. Syphilitic ulceration is not uncommon, and in rare instances tuberculous ulcers are found. These forms usually cause no marked symptoms unless they lead to stenosis, of which syphilis is a prolific cause.

Stricture of the Œsophagus.

Stricture due to the healing of ulcers, usually following traumatic inflammation, is recognized, first, by the gradual development of the symptoms, by the painless nature of the obstruction in the large majority of cases, and by its seat. It is readily found by introducing a bougie; the patient can sometimes localize the area in the upper portion of the œsophagus. The difficulty of deglutition continues over such a long period that nutrition is but slowly interfered with, but gradual emaciation with coincident anæmia develops eventually.

Carcinoma of the Œsophagus.

In cancer of the œsophagus dysphagia is the most prominent symptom. It comes on gradually. The patient expectorates a considerable quantity of frothy mucus, often containing blood, and revealing, on careful examination, cancerous tissue at times. Pain is not generally very severe. Cough is usually present, due to pressure of the cancerous mass on the recurrent laryngeal or pneumogastric nerve. Sometimes the cancer develops in the anterior wall, and ulcerates into the trachea or bronchus. When this complication takes place, the cough is violent. Dyspnoea from pressure is likely to occur. Perforation of the œsophagus into the air-passages is followed by pulmonary abscess or gangrene, or the sudden appearance of dyspnoea, and shortly the onset of aspiration pneumonia. When ulceration causes a pulmonary œsophageal fistula, the condition may simulate that of phthisis.

The difficulty of deglutition due to cancer must be distinguished from that of traumatic or syphilitic stricture and from spasmodic stricture and paralysis of the œsophagus. The history of the case aids in the recognition of traumatic or syphilitic stricture, while the ready passage of a bougie indicates that the difficulty is spasm or paralysis. Cancer usually occurs late in life and is attended with rapid emaciation. Its complications, more common than in other obstructions, are attended with fever and rapid prostration. Cancer may be distinguished from disease outside of the œsophagus by the condition of the stomach beyond the point of stricture. If there is cancer, atrophy is more likely to take place, the change in size being recognized by distending the stomach with air or fluids. The excessive secretion of ropy mucus, salivation included, is, according to Harrison Allen, pathognomonic of disease in the pharyngolarynx or in the œsophagus, at or above the level of the left bronchus. This secretion may be an early indication of cancer of the œsophagus. It may occur in aneurism.

Sarcoma of the œsophagus is very rare. It occurs most frequently in males and presents symptoms like those of carcinoma.

Dilatation of the Œsophagus.¹ Primary dilatation of the œsophagus is an extremely rare affection. The chief symptom is the regurgitation of food, which is neutral or alkaline, and may be returned some time after the act of swallowing. The patient sometimes complains of a sensation of distention along the course of the œsophagus, with heat and burning. The odor of the breath is fetid. If the œsophagus is not deflected, a bougie can be passed.

If the dilatation is secondary, the amount of dysphagia depends upon the obstruction. Food, however, is not returned immediately. After remaining an indefinite time, not longer than two hours, it is regurgitated unchanged. Bougies, of course, do not pass. In sacculated dilatation, which usually takes place in the posterior wall near the pharynx, a bougie may sometimes pass, and at other times may be caught in the sac. The sac may be enlarged, so as to retain a considerable amount of food, which is regurgitated some time after it is swallowed. A sacculated

¹ "Foreign Bodies in the Œsophagus." Allen, New York Med. Jour., Aug. 17, 1895.

diverticulum, from traction on the outside of the œsophagus, may occur when there is disease of the glands of the neck or mediastinum, with adhesions to the œsophagus.

Sacculated diverticulum in the upper portion of the œsophagus is often readily recognized by discovering the alternate presence and absence of a mass when the sac is full or empty.

In the lower part of the œsophagus, the diagnosis between sacculated diverticulum and a simple dilated diverticulum is best made by using Jung's method. His apparatus consists of (1) a small tube which is introduced into the dilated area; (2) a large stomach-tube with small perforations extending well up the sides; and (3) a small stomach-tube which has its openings only at the end. No. 1 is introduced, then No. 2 is passed into the stomach and a small amount of colored fluid poured through No. 1. If the difficulty is a sacculated diverticulum, the fluid will remain in the sac; if it is a dilatation or simple diverticulum, the fluid will pass through the perforations in tube No. 2 into the stomach. If No. 3 is now passed into the stomach through No. 2, the fluid may be siphoned out. The *x-rays* may also be used to demonstrate the nature of a widening of the lumen.

Rupture of the Œsophagus.

This occurs in very rare instances as a result of disease of its walls, or in some instances even from violent vomiting. It causes extreme pain with severe shock. Hemorrhage may occur through the mouth or bowel; pleural effusion is likely to be seen, and the condition soon results in death.

Functional Affections of the Œsophagus.

The functional affections are quite as common as organic disease. They are of longer duration, but are unattended by the same grave effects upon the general system. *Spasm* is one of the most frequent affections. It may be so intense as to lead to temporary stricture. It usually occurs in women. The attack comes on suddenly during the act of swallowing food. The food is at once regurgitated. After subsidence of the perturbation, swallowing can be accomplished, if done slowly. Spasm usually occurs in hysteria. The patient may have had some slight accident in the performance of the ordinary act of deglutition, out of which grew the idea that swallowing can not be accomplished. In consequence, the further acts are performed with trepidation, and slight emotional disturbance at the table may cause a recurrence of the sudden spasm.

Unfortunately, calling attention to the act of swallowing always has the effect of embarrassing the patient, and the taking of a meal under unusual circumstances is sure to be attended by complete dysphagia. Sometimes the idea is conceived that certain forms of food alone can not be swallowed. It is usually thought that solid food gives the distress. Mitchell says that dysphagia occurs early in cases of hysteria; unless relieved, the hysterical symptoms are likely to be transferred to the stomach. I saw a female patient who, after an ordinary choking attack,

for several years could not swallow food in the presence of strangers, or after the slightest emotional disturbance, or if hurried. The spasm disappeared after treatment with bougies.

In *paralysis*, difficulty of deglutition is the main symptom. The course of œsophageal paralysis depends upon its cause. The larynx is usually affected at the same time, so that laryngeal symptoms are present. Paralysis generally comes on very gradually. It may be due to cerebral hemorrhage, tumor, bulbar paralysis, or to general paralysis of the insane. The bougie passes easily, and does not cause irritation. In paralysis there is no regurgitation of food.

CHAPTER IX.

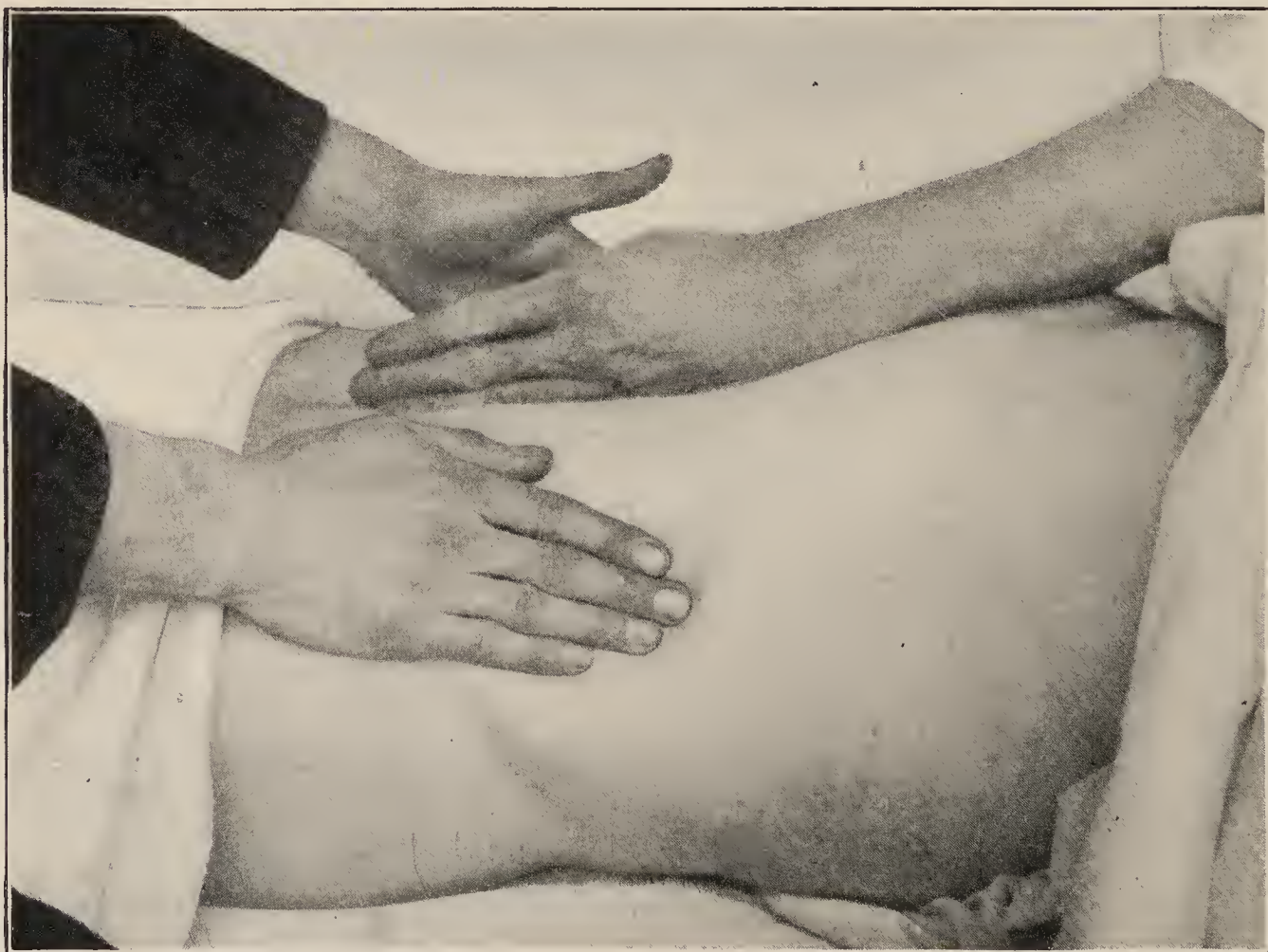
DISEASES OF THE PERITONEUM AND RETROPERITONEAL GLANDS, THE STOMACH AND INTESTINES.

DISEASES OF THE PERITONEUM.

Ascites.

ASCITES is accumulation of fluid in the peritoneal cavity and may be due to general or local causes. The *local causes* are, first, *simple, cancerous*, or *tuberculous* inflammation of the peritoneum; second, portal obstruction from disease of the liver, as cirrhosis, or disease of the portal vein from compression or inflammation; and third, abdominal tumors, which are often attended by ascites, particularly solid tumors of the ovary. The *general causes* of ascites include all those that give rise to dropsy.

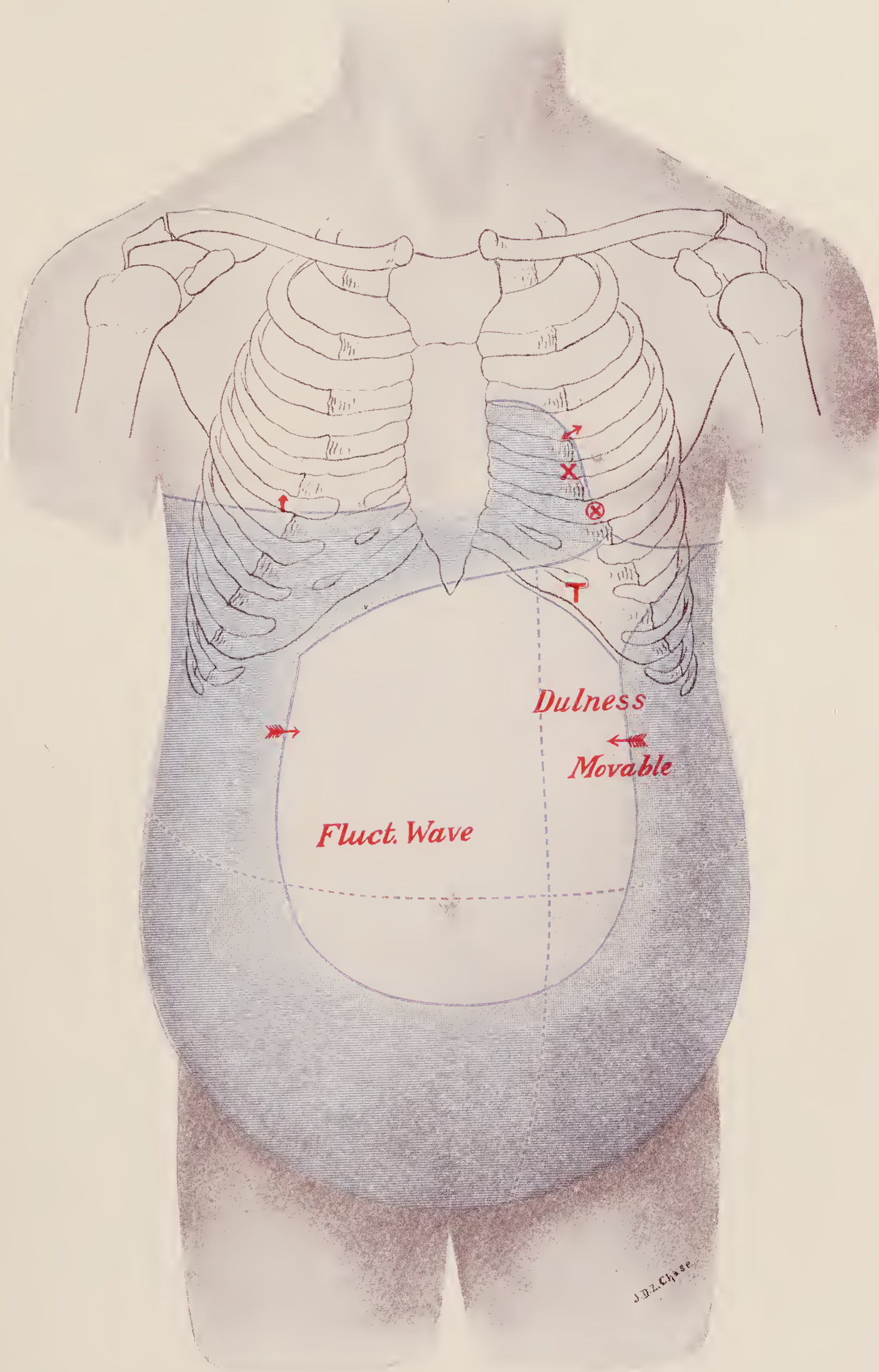
FIG. 360.



Bimanual palpation to determine the presence of fluctuation.

Inspection. (Plate XXXVII.) The surface of the abdomen is usually smooth, but when the effusion is large, the skin becomes tense and *lineæ albicantes* may be present; there may be pouting of the umbilicus. When the ascites is due to disease of the liver or of the portal vein, the

PLATE XXXVIII.



Ascites.

Blue shading shows level of dulness in recumbent posture.
Dotted lines indicate change of level of fluid in other postures.

superficial abdominal veins become enlarged, although such enlargement may be produced by any effusion persisting for a long period of time. When the patient is lying upon his back, the front of the abdomen is flattened and the flanks bulge; when he turns upon his side, the flank that is uppermost becomes hollowed out and the front of the belly prominent. This is the appearance when a moderately large effusion has existed long enough to stretch the lateral abdominal muscles; but when the effusion is enormous, all parts of the belly are distended and the abdomen is barrel-shaped; in such a case the shape is not affected by posture.

Palpation. Fluctuation can usually be detected by applying one hand—the left, for example—firmly against one side of the abdomen while the opposite wall is gently tapped with the middle finger of the other—the right—hand at a point corresponding to the supposed level of the fluid. (Fig. 360.) At first the hand should be placed on the flank, and if fluctuation is not perceived, the hand should be brought forward toward the median line with each successive blow of the percussing finger. Sometimes light percussion will elicit the sign, at others more forcible percussion is required; the faintest tap is often sufficient. Care is necessary not to confound the undulatory vibration of the abdominal walls, produced by percussion, with the wave of true fluctuation; to guard against this error, the former must be cut off by the hand of an assistant placed vertically in the median line. In order to ascertain the position of solid organs in ascites we employ the procedure known as *dipping*, which consists in suddenly dipping down with the tips of the fingers over the organ sought for. The fluid is thus displaced, and the edge or surface of the organ is readily felt.

Percussion. *Dulness* is found over the space occupied by the fluid, which of course corresponds to the dependent portions of the abdomen; sometimes, as Tyson has pointed out, the colon gives rise to tympany in the flanks. In the dorsal position the dulness occupies the flanks and may extend around the lower portion of the abdomen; in the erect posture it may reach to the umbilicus in the median line and to the same level in the midclavicular line.

FIG. 361.



Ascites. Upper limits of dulness indicated by the dotted line. Umbilicus prominent. (Original.)

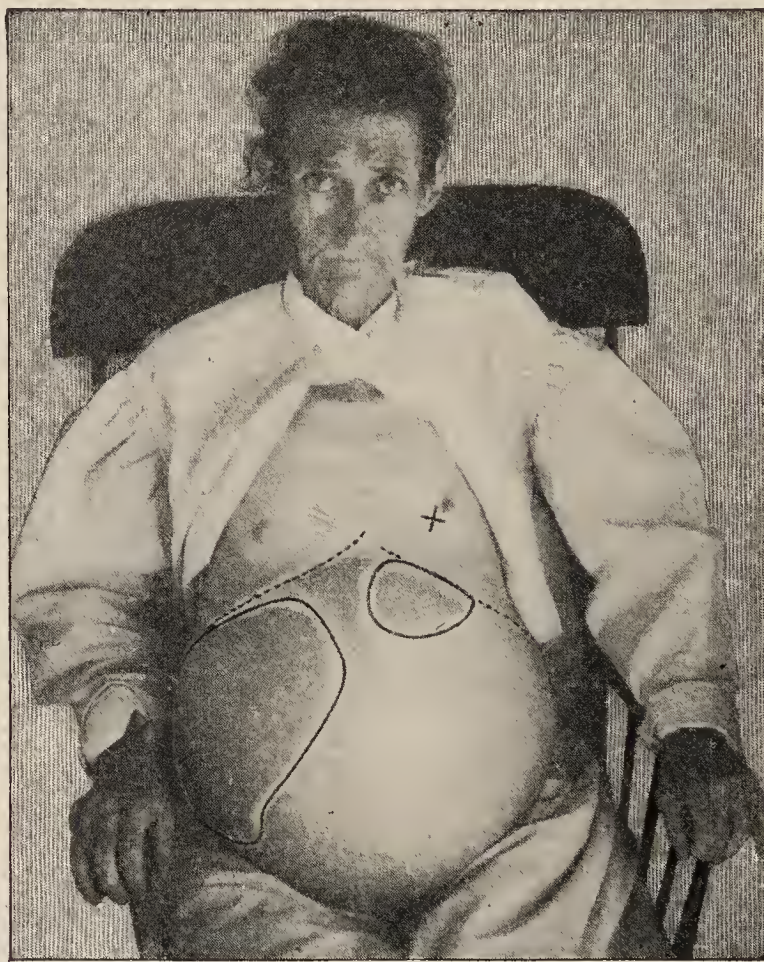
Aspiration. (See Chapter IX., Part I.) It is important to ascertain whether the fluid is *serous* or mixed with *blood* or *chyle*, and this can be done only by aspiration. *Hemorrhagic effusions* are usually found in

cancer and in tuberculosis, although in both of these diseases the fluid may consist of pure serum; in ruptured tubal pregnancy the fluid is always hemorrhagic. Disease of the lymphatics and of the thoracic duct with perforation in rare instances leads to the production of a chylous, milky ascitic transudate. *Chylous ascites* may also be due to the presence of filaria. It should be remembered that *lipæmia*, which sometimes develops as the result of excessive or long-continued ingestion of milk, may be responsible for the turbidity of an effusion into a serous cavity.

The **subjective symptoms** are those due to the primary disease and to mechanical pressure.

Differential Diagnosis. Ascites must be distinguished from enlargement of the abdomen due to *pregnancy*, enlargement due to *ovarian tumor*, and enlargement due to an *over-distended bladder*. During the last month or two of gestation, especially when the woman has previously borne children, the enlargement is general. In ovarian tumor the swelling at

FIG. 362.



Ascites from sarcoma of ovary. Dislocation of liver and spleen. X is apex-beat, not lifted because of fallen abdominal organs. (Original.)

first takes place to the right or left of the median line; when the tumor is very large, the signs may be in the central portion of the abdomen, but the flanks are always tympanitic on percussion. A vaginal examination usually reveals the local disease. A distended bladder should always be thought of, and catheterization performed in doubtful cases. *Pancreatic cysts* may be mistaken for ascites, and large *hydatid cysts* connected with the liver may simulate an accumulation of fluid in the peritoneal cavity. The history and the appearance of the fluid obtained by aspiration point to the correct diagnosis.

Peritonitis.

Acute General Peritonitis. Inflammation of the peritoneum may be acute or chronic, general or localized, primary or secondary. Acute general peritonitis is rarely primary; more commonly it represents a terminal infection in the last stage of chronic Bright's disease or other dyscrasia. A rheumatic form is said to follow exposure to cold, and when the inflammation develops as the result of an injury, the term traumatic peritonitis is used. In the great majority of cases peritonitis is secondary to inflammation in one of the organs covered by the peritoneum or to perforation of an abdominal viscus. Thus, it may follow inflammation of any portion of the gastro-intestinal tract or of the pelvic viscera, or suppurative inflammation of the spleen, liver, or pancreas.

Peritonitis an Infection. In every instance the primary inflammation in the organs mentioned is due to some micro-organism, as the staphylococcus, streptococcus, or *Bacillus coli communis*, and the peritoneal inflammation to subsequent extension of the infection. In perforative peritonitis, as in ulcer of the stomach or bowels, the element of infection also plays the most important part in the process. Inflammation of the gall-bladder may eventuate in perforation with resulting peritonitis; and rupture of an abscess in the liver, spleen, or kidneys, with the evacuation of pus into the peritoneal cavity, is also followed by general peritonitis; but the most common forms are due to appendicitis or to disease of the Fallopian tubes. Acute peritonitis may develop by direct infection in the course of tuberculosis or other systemic infection.

Symptoms. The onset of peritonitis depends in a measure on the cause, that of the perforative form being quite sudden. The disease is ushered in by *chills* and *rigor* with intense abdominal pain, at first localized but rapidly becoming general. The *pain* is constant although subject to exacerbations, and is aggravated by movement and by pressure. The patient assumes the dorsal decubitus with the knees drawn up, in order to relieve the tension of the abdominal muscles. Owing to the pain attending the contractions of the diaphragm the respirations are shallow and hurried, illustrating the superior thoracic type of breathing, and the patients are unable to take a full breath without suffering; while the act of speaking or coughing greatly adds to their distress. The *location of the pain* depends on the seat of the primary infection, which is usually in the right or left lower quadrant where the Fallopian tubes and the appendix are found. The pain of perforating gastric ulcer may be referred to the back, or to the chest or shoulders.

The general symptoms are marked. After the initial chill the *temperature* rises to 104° or 105° F., and then continues at a moderate elevation in ordinary cases. In septic peritonitis, however, the temperature may continue at the initial level or even rise above it, and in rapidly progressing cases a temperature of 105° or 106° F. on the second or third day is not uncommon. The difference between morning and evening temperature is not great except in the case of abscess. The onset of pain and physical signs of distention and rigidity is usually followed at once by *vomiting*, which is painful and at first complete, the vomited matter

consisting of stomach contents, then of yellowish, bile-stained fluid, and finally of greenish material. Later, actual vomiting is replaced by simple regurgitation of fluid, so that the slightest movement or the ingestion of a small amount of fluid is followed by passive regurgitation, unassisted by the action of the diaphragm, of the characteristic greenish-colored fluid, sometimes continuing almost uninterruptedly for from twenty-four to forty-eight hours. The bowels are constipated; they may be loose at first, but *constipation* is characteristic, the intestines being paralyzed from over-distention and from œdema of their walls due to the inflammatory process.

Physical Signs. The *appearance* of the patient at the height of the disease is characteristic: the features are pinched, the eyes sunken, and the expression of the face anxious. (See Expression.) The tissues of the body show the wasting effects of the continual vomiting, and the signs of collapse are marked. The *tongue* at first is furred, but later becomes dry, and often cracked and red. The abdomen is extremely sensitive, so that the patient is unable to bear the weight of clothing or external applications.

Distention gradually develops and the percussion-note becomes tympanitic. The distention may become so great as to push up the diaphragm, causing the above-mentioned superior thoracic type of breathing, and to dislocate the heart so that the apex-beat appears in the fourth interspace. The splenic dulness may be altogether obliterated, and the liver-dulness diminished in size; it has even been asserted that in some instances the latter also becomes obliterated, but recent observations have shown that the obliteration is confined to the anterior aspect of the abdomen; in the axillary region the hepatic dulness, although diminished in extent, persists even when the distention is enormous. The obliteration referred to could only take place in perforative peritonitis. Osler points out that in pneumoperitoneum from perforation the anterior hepatic dulness may be obliterated, although dulness persists in the lateral region on account of the effusion of fluid. When a patient with gas in the peritoneum is turned on the left side, a clear note is heard beneath the seventh and eighth ribs, corresponding to the hepatic region. The abdominal muscles are more or less rigidly contracted.

Spasm of the muscle overlying the primary inflammatory focus takes place at once and is a valuable indication of the source of the infection. In some cases, usually when the inflammation is due to the streptococcus, the abdomen shows but little distention or may even be flattened, with board-like rigidity; the pain is not marked, and tenderness may be absent.

The *pulse* is rapid and feeble and soon becomes thready, ranging from 110 to 150 beats in the minute. During the early stages it may be small and hard—the peculiar wiry pulse of the early stage of peritonitis to which numerous clinicians have called attention.

The *urine* is scanty, and micturition may be frequent and painful, particularly if the inflammation began in the pelvic organs. In suppurative peritonitis the urine usually contains a large quantity of *indican*.

Course and Prognosis. In severe cases death may take place in from

thirty-six to forty-eight hours, but, as a rule, life is prolonged for five or six days, or even longer. The vomiting persists, collapse with falling temperature ensues, the pulse becomes rapid and thready; except when the septicæmia is marked, the mind remains clear throughout the entire attack, and the patient dies of paralysis of the heart. Septicæmia manifests itself by a dusky color of the face, rapid and irregular pulse, slight delirium, dry brown tongue, and other evidences of the typhoid state. In protracted cases some effusion may take place into the peritoneal cavity, and dulness which alters with the position of the patient is noted in the flanks. If recovery takes place, particularly in tuberculous peritonitis, the affection sometimes becomes circumscribed, and in that case the dulness is not movable.

Diagnosis. It is essential in making a diagnosis to ascertain, if possible, the primary source of the infection or inflammation. To determine this, we inquire into the age, sex, and history of previous disease. In young male adults appendicitis is first to be thought of; in females, inflammation of the pelvic organs. In chlorotic subjects, if the pain is high up, a history of ulcer of the stomach must be inquired for. Later in life, particularly if there has been jaundice, the possible history of frequent attacks of gallstones and of hepatic disturbances must be ascertained. All forms of intestinal obstruction must be sought for. Frequently, however, a definite cause can not be ascertained. Peritonitis occurring in the course of typhoid fever is usually due to perforation, and its recognition, as a rule, presents no difficulties; but the mental state of the patient may be such that he does not complain of pain. In such cases local tenderness, rigidity, and the sudden occurrence of a leucocytosis are almost distinctive signs.

The most important conditions to be borne in mind are: *enterocolitis*, *acute hemorrhagic pancreatitis*, *intestinal obstruction*, and *rheumatism of the abdominal muscles*.

The distinction of acute peritonitis from *enterocolitis* is usually not difficult if due attention is paid to the development of the case. The pain is less severe and more colicky in enterocolitis; the general tenderness is not so great as in peritonitis, and the distention does not interfere with respiration to such a marked degree. Diarrhœa is more common in enterocolitis; collapse, if present, is less pronounced.

Acute hemorrhagic pancreatitis may simulate peritonitis by the sudden onset and the intensity of the pain and by the occurrence of shock.

The diagnosis from *obstruction of the bowel* is difficult in the absence of a distinct history; but in peritonitis stercoraceous vomiting does not occur until late, and the tympanites and pain are more general. Peritonitis frequently accompanies or follows intestinal obstruction. A tumor, if one be present, may point to the true nature of the case; or a discharge from the rectum may reveal invagination as the exciting cause.

Rheumatism of the Abdominal Walls. There is no history of sudden acute pain followed by general pain; the fever is less marked; the respirations are not interfered with, the pulse is not so rapid, and symptoms of collapse do not supervene. A rheumatic pharyngitis or inflammation of muscles in some other portion of the body may occur simultaneously.

Peritonitis is simulated by a condition to which the name *hysterical peritonitis* has been applied. It occurs in hysterical subjects, and every feature of the true form is imitated. The mode of onset, the decubitus, the difficulty in micturition, and the local distention and tenderness of the abdomen are characteristic of both. In a few cases that I have seen the vomiting differed from that of true peritonitis, both in the mode of ejection and in the character of the fluid. It must not be forgotten that the temperature may even be elevated, and collapse may take place in the hysterical form. In the cases that I have seen, the abdominal facies did not develop; on the contrary, the facies of hysteria, with the self-interest

FIG. 363.



Bimanual palpation to determine if pus is localized in the retrocaecal region.

exhibited by the patient, and the precision with which symptoms were narrated, coupled with emotional or other manifestations of hysteria, suggested the true nature of the affection. The case is judged by the history of other manifestations of hysteria and the presence of permanent stigmata of the disease; there is always a positive absence of cause and of disease in any of the abdominal viscera. Sometimes if the patient's attention is diverted, the tenderness on pressure may not be complained of. I am not familiar with the results of examination of the urine in this form of peritonitis; indican should not necessarily be increased, as it is found to be in acute suppurative peritonitis.

Local Circumscribed Peritonitis. The causes of localized peritonitis are those of general peritonitis—extension of inflammation from, or perforation of neighboring viscera. After perforation has taken place the process remains localized if the inflammation progresses rapidly enough to bring about immediate isolation of the perforated area from the gen-

eral cavity of the peritoneum. Accordingly the regions of predilection for the development of local or circumscribed peritonitis, with or without perforation, are the same as those that have been indicated as the usual starting-point of general peritonitis. But although the inflammatory focus may be retained by a limiting wall, the process may nevertheless, after suppuration has begun, undergo gradual extension and the pus may burrow in various directions.

Circumscribed peritonitis in the lower half of the abdomen is due to disease of the vermiform appendix or of the Fallopian tubes; the local signs are, first, those of pain and tenderness; second, the development of tumor.

Subdiaphragmatic Abscess. Subdiaphragmatic or perigastric abscess is a collection of pus walled in by the stomach, spleen, diaphragm, colon, and the abdominal walls, and may be associated with local peritonitis in the upper half of the abdomen. The most common cause is irritation from a gastric ulcer that has nearly or quite perforated and has formed adhesions with the surrounding viscera. This was the cause in 41 out of 52 cases analyzed by Fenwick, while in 6 it was associated with cancer, and in 4 with abscess commencing externally. *Pain* in the epigastrium was the most prominent symptom; some of the patients were dyspeptic, and vomiting occurred also; while, singularly enough, hæmatemesis is mentioned in only 2 cases. *Fever* is quite marked. Fenwick thinks that in every case of perigastric abscess, except in persons affected with phthisis, cancer, or some other chronic exhausting malady, the first formation of the abscess will be accompanied either by collapse and signs of general peritonitis, or by sudden and severe pain in the epigastrium with indications of local peritonitis.

The *physical signs* are not pronounced; a tumor, according to the same author, is rarely distinguishable except when the condition is caused by cancer. The percussion-note, when a tumor is present, is at first dull but later becomes tympanitic, and the tumor is not movable on inspiration or external pressure; there may, however, be arching outward of the ribs. Palpation is difficult on account of the tension of the abdominal muscles. The degree of displacement of surrounding viscera depends on the size of the abscess and the extent of the adhesions. The breathing is embarrassed by the upward pressure of the diaphragm against the lung and heart, and the dulness may extend as high as the angle of the scapula, simulating a pleural effusion. It should be borne in mind in this connection that the lower limit of the lungs in health corresponds to the eighth or ninth interspace. There may also be dulness in the axillary region; and if the abscess is on the right side, it may simulate an enlarged liver and cause a considerable extension of the hepatic dulness anteriorly, laterally, or posteriorly. If the local peritonitis in the epigastric region is secondary to disease of the pancreas, it may be limited to the lesser peritoneum and cause the physical signs of effusion into that cavity (see Diseases of the Pancreas); while subdiaphragmatic abscess is not limited to the lesser peritoneum and must be recognized by the history of a previous lesion capable of causing perforation and by the general symptoms of abscess.

Subphrenic Pyopneumothorax. Sometimes gas is formed in con-

nection with the abscess, and amphoric sounds are heard on auscultation and percussion both in the abdomen and in the thorax. In this condition, to which the name *pyopneumothorax subphrenicus* has been given, the abdomen becomes tense, tender, prominent, and tympanitic on percussion (see page 918). It must be distinguished from *left-sided pneumothorax*, which pushes the left wing of the diaphragm down and therefore increases the area of dullness, while the left lobe of the liver and the spleen become palpable. In subphrenic pyopneumothorax, on the contrary, the liver and spleen are not palpable, nor can their limits be outlined by percussion. In left pneumothorax the heart is dislocated to the right and the cardiac area is tympanitic on percussion instead of dull, while the impulse is seen in the epigastrium or even to the right of the sternum; in subphrenic pyopneumothorax, on the other hand, the heart is displaced upward and the impulse appears in the nipple-line, while only the lower half of the cardiac area is tympanitic. Pyopneumothorax subphrenicus must not be mistaken for *dilatation of the stomach*.

Chronic Peritonitis. Symptoms of chronic diffuse peritonitis may follow an acute attack, or may occur in the course of tuberculosis. The intestines and peritoneum are matted together. General pain and tenderness, with a prolonged period of ill health, attend the diffuse form. (See Tuberculous Peritonitis.) If there is considerable fibrous proliferation, even though not cancerous or tuberculous, the abdomen becomes retracted, the muscles rigid, the note over the abdomen modified or dull tympanitic, especially in the upper half of the abdomen and over the liver. Sometimes a fremitus can be felt. The patients are under weight and without strength. The pain may continue a long time. The disease finally results, at least clinically, in such compensation that the patient is able to continue his usual occupation. Owing to the formation of adhesions the patients have painful sensations of a dragging character, the significance of which is not serious, however, unless, as has been seen in intestinal obstruction, coils of intestine become caught in these adhesions.

Cancer of the Peritoneum.

Cancer of the peritoneum is a disease of old age and occurs more frequently among women than among men. Although sometimes primary, in the majority of cases it follows cancer in other organs, as the stomach, liver, or uterus. The indurated and nodular omentum forms an irregular mass lying transversely across the upper zone of the abdomen and may be painful on pressure. A tumor of the same physical characters is seen in tuberculous peritonitis, and I have seen several such tumors in the aged without apparent cause, unless it were proliferative peritonitis. (See Tumor.) Ascites is usually present and the enlargement of the abdomen is in striking contrast to the emaciation of the rest of the body; the ascitic fluid is bloody. Sometimes pain is the most prominent symptom. Progressive emaciation, chronic ascites without cause, and a localized tumor without the occurrence of fever are strongly suggestive of carcinoma; and if these symptoms are present without signs of cancer in some

other organ, as the stomach, rectum, or uterus, there is probably primary cancer of the peritoneum.

Retroperitoneal sarcoma, or Lobstein's cancer, may be central or lateral; The growth is very large, deep-seated, and usually fixed; it can be detected above the sacrum by rectal examination; ascites and the general symptoms of cancer are present. The growth is crossed in front by the intestines, which transmit to the palpating hand a peculiar sensation like the fremitus of a hydatid cyst, as in Barrow's case. Lockwood's cases were thought to be solid ovarian tumors. Sarcoma of the glands and tissues in the retroperitoneal space, according to J. Dutton Steele, is slightly more common in males than in females, and more common in the first decade and after the fiftieth year. The duration is about nine months. Of the 65 cases collected by Steele, 39 per cent. were spindle-cell sarcoma, 34 per cent. round-cell sarcoma, 14 per cent. lympho-sarcoma, and 13 per cent. were mixed cases. The growth originates in the lymph-glands or in fibrous connective tissue surrounding the kidneys, the spinal column, the pelvis, or the sheaths of bloodvessels. The onset is insidious, the first appreciable symptoms being the presence of a tumor, or the effects of pressure upon the vessels, nerves, or viscera of the abdominal cavity, the nature of which depends upon the site of the tumor; varicocele is a frequent concomitant of the disease. It is often impossible to distinguish retroperitoneal sarcoma from malignant disease of the kidney or of the suprarenal bodies. The diagnostic features are (*a*) the rapid growth; (*b*) the position of the colon, which is pushed in front of the tumor, particularly if it is lateral; (*c*) the pressure-symptoms; (*d*) movability of the tumor both with respiration and independently, and sometimes fluctuation.

Tuberculosis of the Peritoneum.

The tuberculous process in the peritoneum may be acute or chronic. While the symptoms are often acute and alarming, in the larger proportion of cases the process is more *chronic* and is attended by characteristic local, and by general symptoms resembling those of typhoid fever—indeed, a true typhoid state may develop in the course of the disease. In rare instances the malady runs its course without producing any symptoms either local or general. In protracted and moderately severe cases the *fever* is moderate and may be continued or remitting in type; in old people the temperature is frequently subnormal. (See Fig. 366.) In more severe cases the fever is high, but always irregular in type, although the latter may also approach the remittent form. Profuse sweating is characteristic of the disease, and the emaciation is more or less rapid.

The Local Symptoms. The cases may be divided into four classes, according as they exhibit: (1) abdominal enlargement with effusion; (2) enlargement with tumors; (3) a combination of the two; or (4) enlargement without evidence of fluid or tumor in the abdomen. In the forms included in the second and fourth subdivisions the abdomen may subsequently undergo retraction.

1. **Enlargement with Effusion.** The local symptoms and the physical signs are those of ascites ; but the distention is never so great as in ascites from cirrhosis of the liver. Often the fluid is confined by adhesions in the right or left quadrant of the abdomen, in which situation fulness and fluctuation may be readily detected.

2. **Enlargement with Tumors.** (Plate XXXIX.) The tumors are usually found in the upper zone of the abdomen, and may be localized in either quadrant, or extend from the right to the left. They are usually due to tuberculosis of the omentum, with secondary contraction. In some instances an indurated tumor, somewhat tender on pressure, may

FIG. 364.

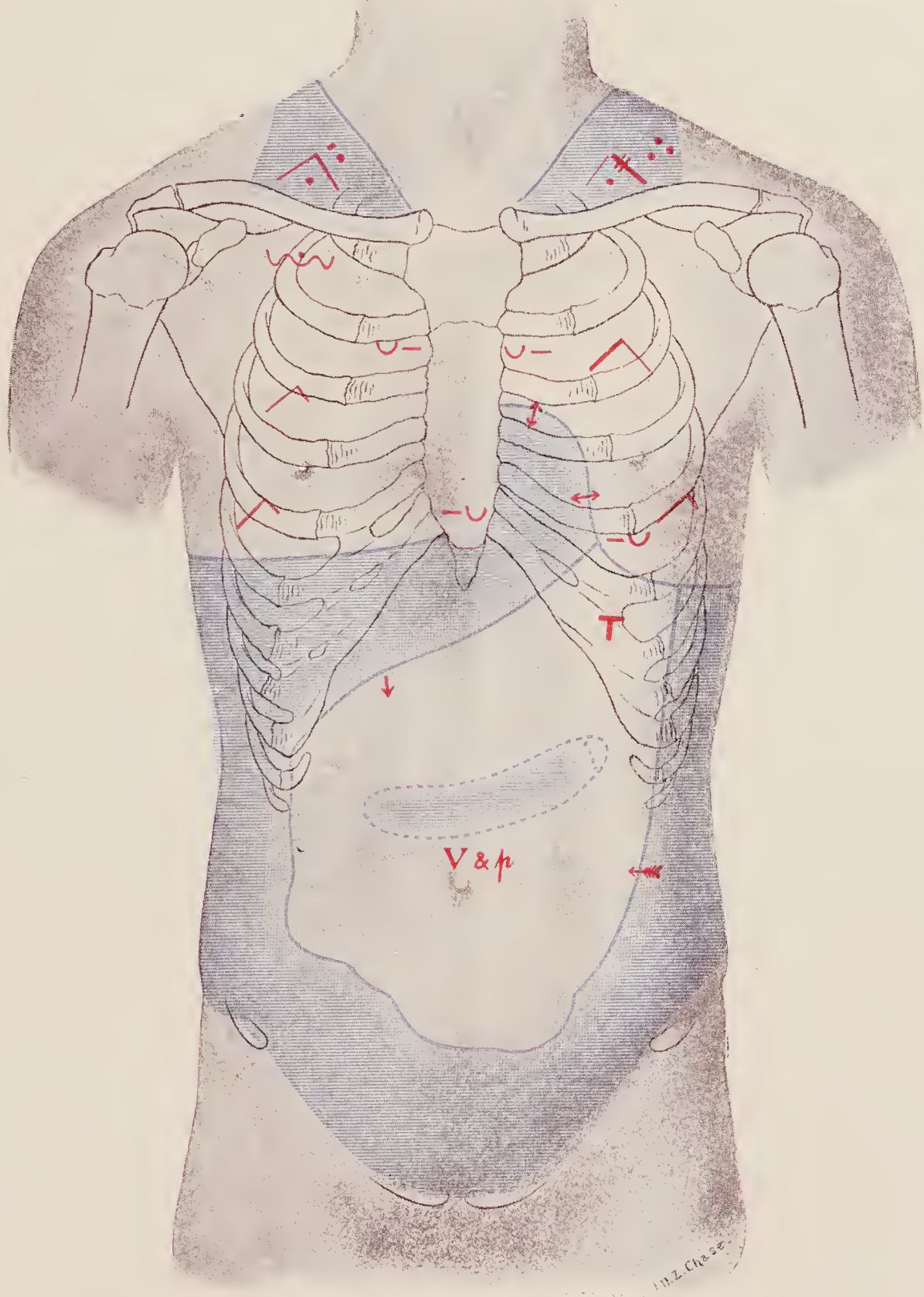


Tuberculous peritonitis; pulmonary tuberculosis. + site of cardiac impulse. (Original.)

extend across the abdomen midway between the xiphoid cartilage and the umbilicus, or at the level of the umbilicus, varying from two to four inches in width and sometimes continuous with the liver-dulness. In other instances more distinctly localized masses may be felt either to the right or to the left of the umbilicus. They may be movable and vary with the position of the patient. I have never seen tuberculous masses in the lower quadrants. In children with *tabes mesenterica* the tumors may be made out close to the vertebral column in the median line, extending to the brim of the pelvis, although at the lower portion they are not so distinct. The intensity of the dulness over the tumors depends upon their relation to the bowels and the degree of intestinal distention. Instead of dulness a modified tympany or muffled resonance may be observed.

3. **Cases in which Effusion and Tumors are Present at the Same Time.**

PLATE XXXIX.



Tuberculosis of the Peritoneum.

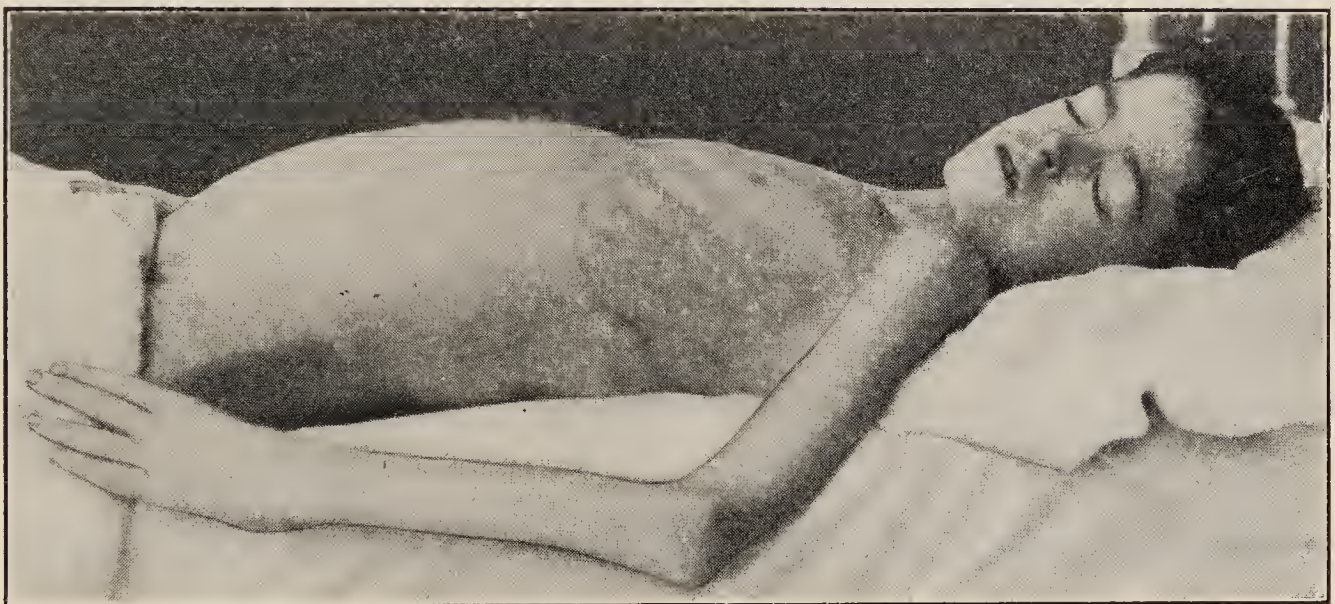
Abdominal exudate (not freely movable); omental tumor. Consolidation at apices.

These present symptoms common to the two conditions, although the tumors are not so distinctly defined.

4. Absence of Effusion and Tumors. When effusion and tumors are not present, the increased thickness of the peritoneum and of the intestinal walls produces a modified dulness over the entire abdomen. When retraction takes place, the resonance is of a woodeny character, the abdomen is more or less tender, and an ill-defined induration to which the term *carreau* is applied may be present.

The only other distinctive symptoms, apart from those described, are the symptoms, if any be present, of tuberculosis in other situations. The appetite is usually impaired, there is some atonic dyspepsia, vomiting may occur at regular intervals, and the bowels may be constipated, although in my experience they have usually been relaxed. Anæmia develops, and the skin becomes harsh and dry; the emaciation is often extreme, and the body covered with eruptions and boils; œdema is sometimes present over the ankles. Death results from exhaustion and from tuberculosis in other localities.

FIG. 365.



Tuberculous peritonitis. Note the emaciation, the length of the arm, the enlargement of the wrist, the size of the hand, and the tapering fingers.

Diagnosis. Cases belonging to the first and fourth classes above mentioned probably present the greatest difficulties. Age is an important factor; peritoneal tumors, with or without effusion, in young subjects are almost always due to tuberculosis; in the aged they must be distinguished from carcinoma or chronic peritonitis from other causes. The existence of diarrhœa is rather against carcinoma. In males the primary lesion often develops in the testicles. Sacculated peritoneal effusions may be confounded with ovarian or other abdominal tumors, especially if the tubercles develop primarily in the tubes or uterus. In a recent case the autopsy disclosed a large caseating ulcer inside the uterus, with tuberculous disease of the Fallopian tubes and of the peritoneum. The right tube was chiefly affected. The effusion during life was encysted and confined to the right lower quadrant, was not movable with the patient, and fluctuation could be made out both by external palpation and on bimanual examination *per vaginam*. The fact that the normal resonance in the flank was replaced by dulness was the only evidence that the

condition was not a growth. As Osler has pointed out, the association with salpingitis must arouse suspicion, particularly if at the same time signs of tuberculosis are found in some other part of the body, as the apex of the lungs or the pleura. The history of the case and the irregular development of the disease associated with gastro-intestinal disturbance rather than disturbance of uterine function are points in favor of tuberculosis.

The diagnosis may be obscured by the presence of tympanites, which is of frequent occurrence.

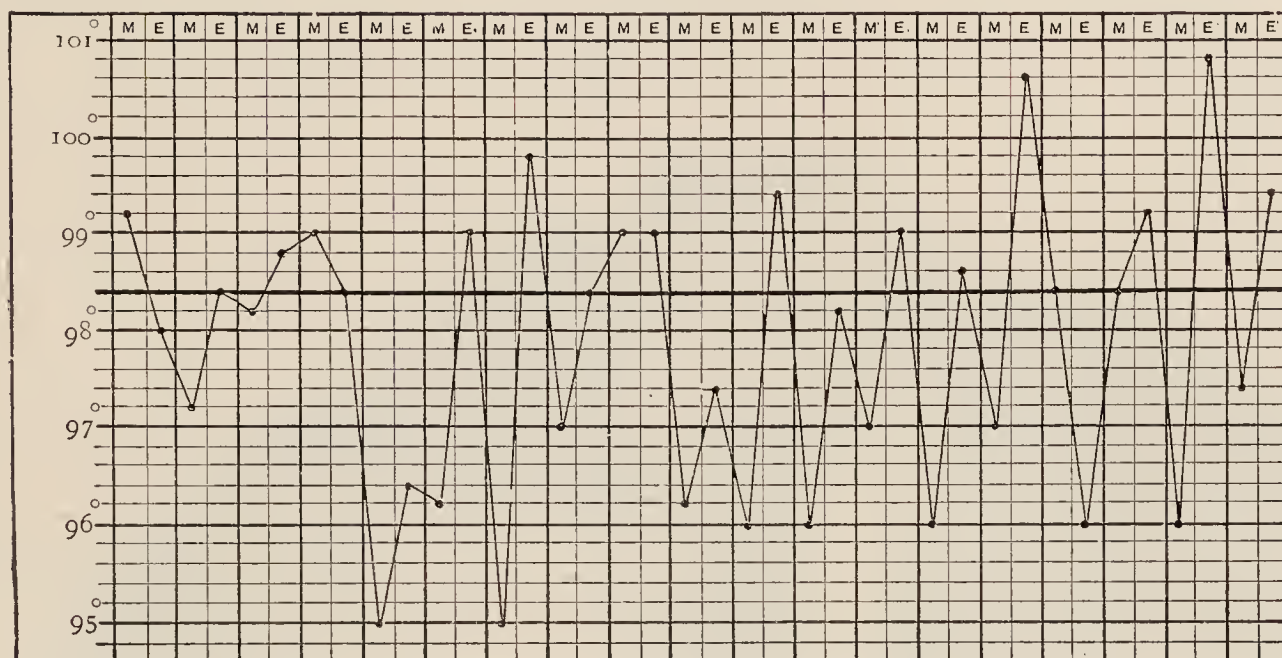
In not a few instances the local physical signs are apparently due to perihepatitis. In the case of a child under my care the local signs during life were of this character, and the symptoms were simply loss of appetite, with discomfort, weight, and fulness below the sternum. Both the right and left lobes of the liver were covered with enormously thickened tuberculous peritoneum, while the lower portion of the abdomen was the seat of simple plastic peritonitis.

Suppurative peritonitis may be closely simulated by acute tuberculous peritonitis, although usually the course of the latter is more prolonged and the fluctuations of temperature are less pronounced; otherwise it can not be distinguished from acute general peritonitis save by the absence of any of the recognized causes of that condition. A history of exposure to tuberculous infection, or the presence of tuberculosis in some other portion of the body, may be of service in determining the nature of a given case; thus associate tuberculosis of other serous membranes, as the pleura or pericardium, often develops in a short time an associated process that does not take place in ordinary peritonitis. There is diarrhoea in most cases—at least it has been present in the few instances that I have seen of this form of tuberculosis. Nevertheless, the diagnosis is sometimes impossible. Henry has called renewed attention to the occurrence of inflammation about the navel as a sign of tuberculous peritonitis; he believes the peri-umbilical erythema is pathognomonic of the affection.

Appendicitis. Acute tuberculosis of the peritoneum may also simulate appendicitis, first, in the local symptoms and physical signs; and, second, in the occurrence of subsequent infection of the peritoneum. In acute tuberculous appendicitis, however, the signs of a tumor are not so marked as in true appendicitis; although in one instance Keen operated upon a patient of mine, a healthy laborer in a rolling-mill, who had the classical symptoms of appendicitis. At the operation the appendix was found to be perforated and hanging in an encapsulated abscess. A fecal fistula ensued which did not heal, and within two months the patient died of general tuberculosis. The appendix was the seat of primary tuberculous ulceration. In another instance the appendicitis arose in the course of tuberculosis. In a third instance the patient, aged forty-five years, was admitted to my wards in the Philadelphia Hospital with high fever and pain in the abdomen, at first more marked along the margin of the liver. By the end of twenty-four hours the pain became more intense in the right lower quadrant of the abdomen; tenderness at McBurney's point was distinct; the painful area was enlarged and dull on percussion, and

the surface slightly œdematous. Fluctuation could not be detected. Extension of the leg was painful. Rapid general peritonitis ensued during which the surgeon saw him, but declined to operate until the subsidence of the attack. When the attack subsided, the local signs of tumor were not present. The fever persisted irregularly for a short time, while

FIG. 366.



Tuberculous peritonitis. Subnormal temperature. (Original.)

the more acute peritoneal symptoms subsided; then the right pleura became infected, and cough ensued with expectoration of mucopurulent fluid, which, however, did not contain tubercle bacilli. Subsequently the left pleura and the pericardium became involved. The entire course of the disease was marked by diarrhœa, profuse sweats, rapid emaciation, and exhaustion. Death took place at the end of five weeks, and at the autopsy general serous tuberculosis was found.

THE STOMACH.

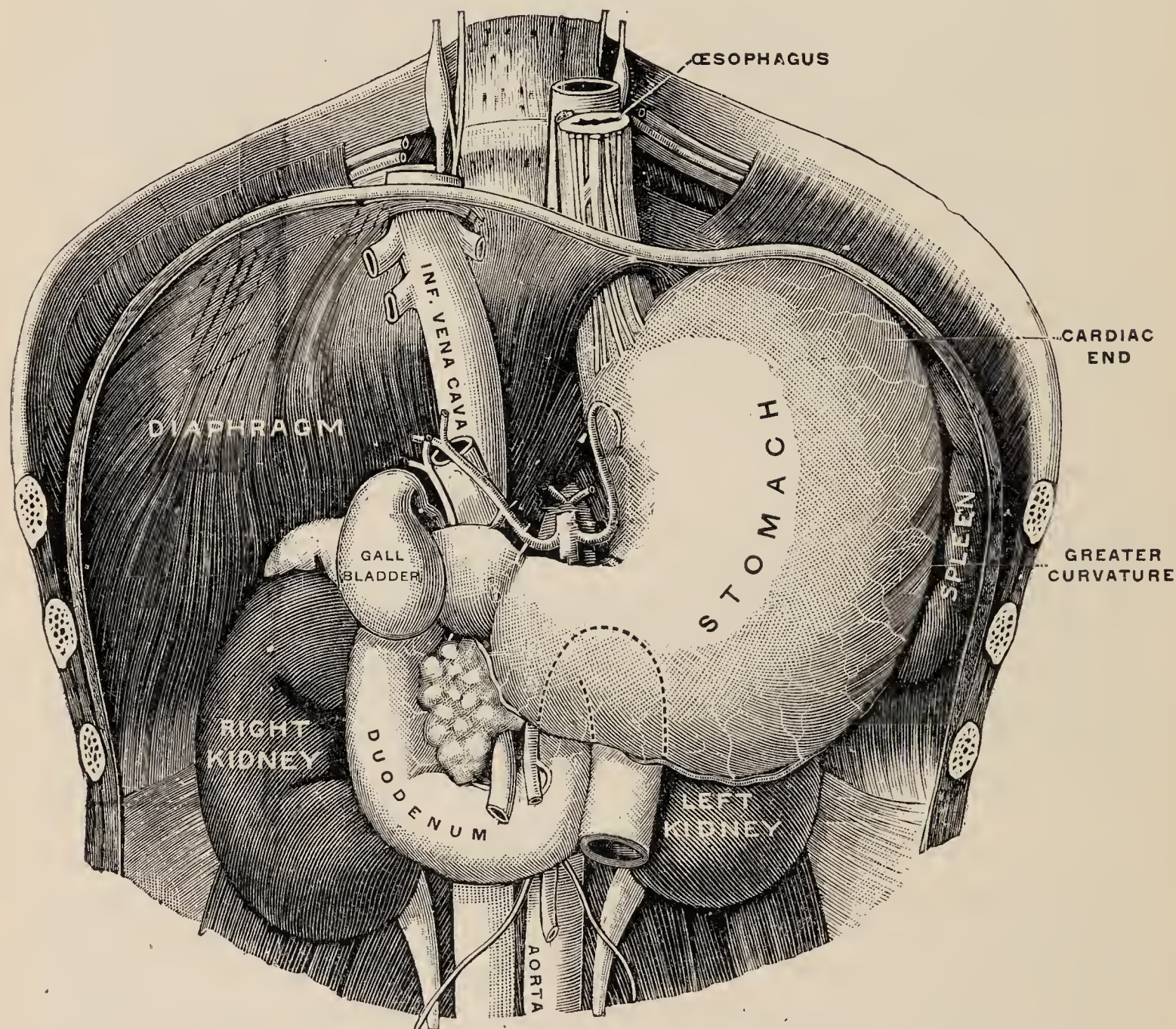
The stomach is a canal in which the food is detained for the purpose of solution and digestion. Its walls are made up of mucous membrane, muscle, and peritoneum. It is richly supplied with bloodvessels. Because of its great functional activity it has an abundant nerve-supply. It is, moreover, surrounded by rich plexuses of sympathetic nerves, through which and its special nerve, the pneumogastric, it is in intimate relation with every organ of the body.

The Symptomatology. The local symptoms of disease of the stomach are dependent upon: (1) the morbid process; (2) the effect of the process upon the anatomical structure and size of the organ (atrophy, dilatation, tumor); (3) the effect upon its function.

1. The Morbid Process. The symptoms due to the *morbid process* are not different from the symptoms of similar morbid processes elsewhere, save that they are modified by the function of the organ or its special construction. Hence, congestions are attended by discharge of mucus; inflammations by pain and by a flow of mucus and pus; ulcers

by pain and the accidents of ulceration (hemorrhage); malignant disease by pain and swelling (tumor), and its accidents, hemorrhage and obstruction; while each condition is attended by characteristic general phenomena. But the stomach is highly sensitive and resents the intrusion of disease or of that which (1) causes disease or (2) irritates the affected part. Expression of this resentment is shown in hyperæsthetic symptoms—*pain*; in the abolition or derangement of function—*indigestion*; and in the great pathological reflex act of the stomach—*vomiting*.

FIG. 367.



Stomach and duodenum, the liver and most of the intestines having been removed. The pyloric end of the stomach should be conceived as turned directly backward. (TESTUT.)

It will be seen later that this may be a symptom of any local morbid process affecting the organ, either directly by the disease which is its exciting cause, both of which are operative in irritant inflammations; or indirectly because the process has set up undue sensitiveness. In the latter instance any such material as food, which the stomach is accustomed to receive, becomes as much an irritant as mucus, pus, or blood.

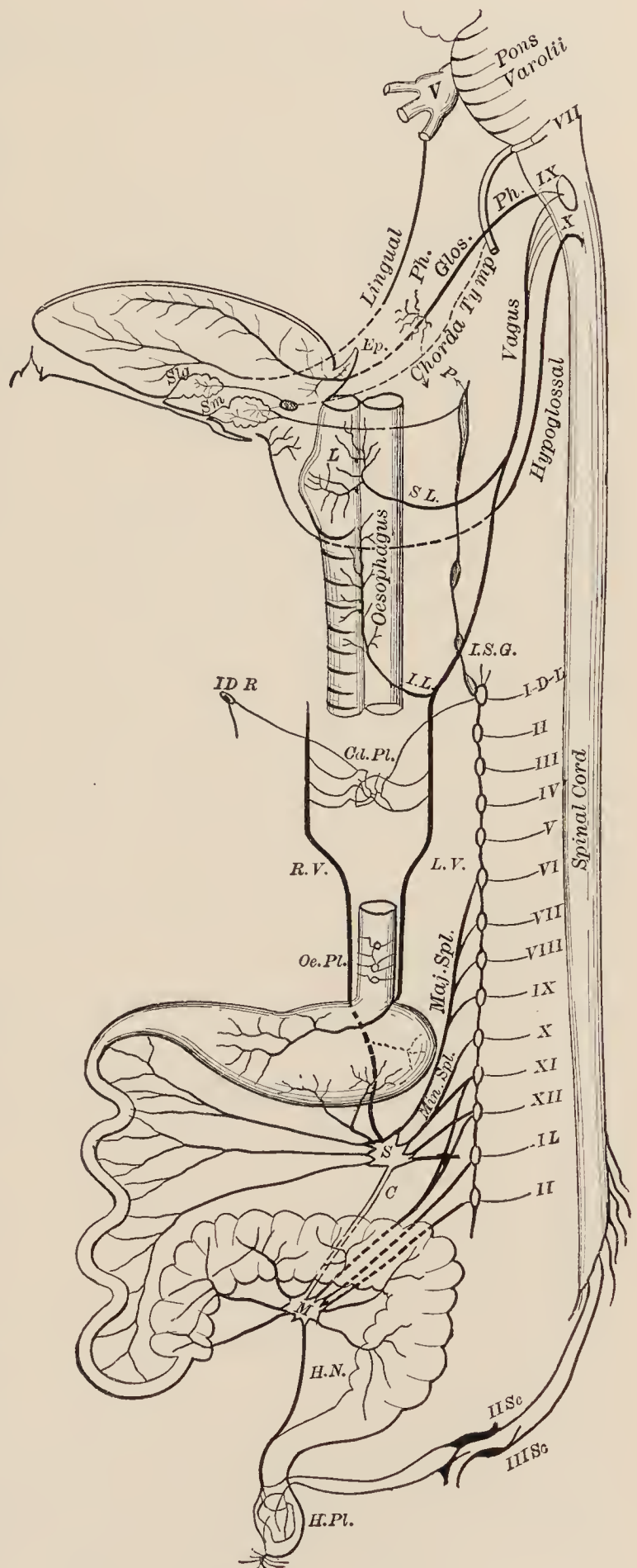
2. Anatomical Symptoms. The morbid processes modify the anatomical structure and lead to other morbid conditions, as we see when dilatation succeeds inflammation or obstruction at the orifices. The symp-

toms of the secondary conditions are the same as elsewhere—in atrophy, diminution in size; in dilatation, increase in size, with retention and fermentation, and finally discharge of the contents by vomiting.

NERVE MECHANISM. In the study of the symptomatology of gastric diseases the anatomical relation of the organ through its vascular and nervous connections must be considered. The student is sufficiently familiar with physiology and pathology to know that each organ has a representative in the central nerve-mass, the brain, and that disease in one organ will influence the function of, and create morbid symptoms in another organ with which it has intimate nervous connections.

The central representative or centre is influential in proportion to the power and activity of its peripheral adjunct, and is, moreover, influenced by the higher, psychical centres, which it in turn modifies. It influences or modifies lower centres—(1) functional, (2) vasomotor, (3) motor, and (4) sensory. The result of this mechanism in the case of the stomach is: 1. That functional alteration or organic disease of (a) the gastric centre, or (b) of centres of higher control, or (c) of the nerve that connects the centre and the organ—pneumogastric nerve—produces gastric symptoms. 2. That gastric diseases produce reflex symptoms in other organs, as cardiac palpitation. 3. That disease of other organs produces gastric symptoms or disease; instance the vomiting of pregnancy, of renal calculus, or of disease of the testicle, and the gastritis of kidney disease. Thus vomiting is caused by emotion (higher centre) influencing the pneumogastric (lower centre); by a tumor pressing upon or destroying the pneumogastric centre; or by a tumor, such as an aneurism, pressing on the pneumogastric

FIG. 368.



Showing the innervation of the digestive system.

nerve. I have taken the simplest illustrations. When we come to the study of gastric neuroses, the extraordinary influence of the nervous mechanism will be appreciated; and when hysteria is studied, the physiology of its extreme gastric symptoms will be recognized. When the mechanism and clinical course of vomiting are studied, it will be found among other causes to be frequently due to affections of the blood, the poisons of which irritate either cerebral centres or nerve-plexuses in the stomach.

VASCULAR MECHANISM. Gastric diseases also arise because of some disturbance in the *vascular supply*. Thus in heart disease with venous stasis the gastric veins become the seat of congestion, with consequent gastric catarrh; and hepatic disease causes portal congestion and gastric catarrh.

3. Functional Symptoms. Any local disease of the stomach must influence its function; therefore, conversely, functional symptoms must be present in all local diseases. But functional disorder may be present without local anatomical change; the impairment is nearly always induced through the influences of the nervous system. The functions of the stomach are to digest and to absorb some of the products of digestion. The digestive function is both motor and chemical, and its completeness depends upon the food being thoroughly mixed with and dissolved in the gastric juice. The symptoms, therefore, must be due to changes (1) in the motor, (2) in the secretory, and (3) in the absorptive functions of the organ. The gastric functions may be increased or diminished; the former are primary and usually temporary aberrations; the latter succeed the former and are permanent. The functional symptoms, therefore, are the symptoms of what we know as *indigestion* or *dyspepsia*. They are described in the account of the subjective symptoms and also in the section on Gastric Neuroses.

4. Toxic Symptoms. The toxic symptoms arising in gastric disease are worthy of a few words. They are nervous symptoms due to the absorption of ptomaines or products of imperfect assimilation. If absorption of the toxins takes place suddenly and in large amounts, coma and convulsions occur (Kussmaul's symptom); if gradually, hypochondriasis, melancholia, mental depression, and with these symptoms vasomotor phenomena of various kinds arise.

It follows, therefore, that in unravelling the symptomatology of gastric disease we must first note: (A) The *subjective symptoms* due to (1) to morbid processes, (2) to alterations of function, (3) to alterations of size (sense of fulness, etc.). (B) The *objective symptoms* due (1) to morbid processes, (2) to alterations of function, (3) to alterations of size.

Diagnosis from Disease of Contiguous Organs Functionally Related. The student will soon learn that functional diseases of the stomach can not be readily differentiated from diseases in other organs functionally related. He will find that to draw hard-and-fast lines between gastric and intestinal indigestion, or between so-called disordered gastric and hepatic function, is generally impossible. Organs that are closely related in physiological function, and have a common nerve- and blood-supply, can not be dealt with separately in conditions of disordered function.

Hence, indigestion and biliousness, or simple acute gastritis and duodenitis, are beyond the pale of close discrimination—in fact, the symptoms of each blend in a manner.

From what has been written above, it may be inferred that in order to judge correctly of the nature of gastric lesions we must ascertain (1) whether the gastric symptoms are dependent upon disease of other organs—particularly the eye, nose, and genitalia, the heart and kidneys—by an examination of each organ; and (2) whether other symptoms that may be present are caused by the gastric disease.

The Stomach in Other Diseases. Diseases of the stomach frequently mask other diseases; in other words, patients will complain of gastric symptoms that are but concomitant phenomena, behind which there are graver conditions. Thus in disease of the kidney, in phthisis, in chronic bronchitis, in emphysema, in valvular disease of the heart, catarrh of the mucous membrane of the stomach is of frequent occurrence, and is entirely due to the primary disease.

Tuberculosis. In tuberculosis the local gastric symptoms are often the most prominent feature. Thus in the earlier stages of phthisis loss of appetite and vomiting are of constant occurrence. The dyspeptic symptoms in a large number of cases precede the pulmonary symptoms, and may be so pronounced as to mask them entirely. The patients are usually delicate and anæmic; they complain of loss of appetite and mild indigestion, and are feeble and languid; there is some regurgitation of food. The case is treated as chronic catarrhal gastritis, but does not improve. On examining the lungs the physician is surprised to find a small area of consolidation, and upon inquiry may find that subjective symptoms of tuberculosis have been present for a considerable time. Every practitioner has seen scores of phthisical patients who, even when the disease is far advanced, believe that their symptoms are entirely due to disorder of the stomach. In addition to the early catarrh that precedes tuberculosis, symptoms of other gastric disease may occur. The well-known association of simple ulcer and phthisis is familiar; both occur at the same time of life, yet the presence of gastric symptoms may prevent investigation into those of pulmonary origin.

Anæmia and Chlorosis. In anæmia and chlorosis changes in the digestive tract are common. The functions of the stomach are impaired by the general blood condition. Here, too, we frequently have the association of ulcer with the general condition, although the danger of overlooking either is not so great as in tuberculosis.

Valvular Affections of the Heart. In valvular affections of the heart chronic catarrh of the stomach may result from venous congestion. As the symptoms may point to the gastric condition alone, it is necessary in all cases of chronic gastric catarrh to examine carefully into the condition of the heart. Over and over again patients apply for treatment, not on account of cardiac symptoms, but because of gastric disorder. They will be treated in vain if the primary cardiac affection is not discovered. Many cases of gastric catarrh have been cured by the use of digitalis.

Disease of the Kidneys. In disease of the kidneys the stomach is frequently involved. Vomiting and other symptoms of gastric disorder

may occur long before dropsy or any objective sign that would lead to a correct diagnosis. The gastric symptoms are due to chronic uræmia. Gastric symptoms also occur in other conditions of the genito-urinary tract, particularly in long-standing retention from chronic obstruction. Renal tumors may cause only disturbances of digestion, while gastric symptoms due to movable kidney are well known. The symptoms in the latter condition arise, first, from mechanical causes, as pressure of the kidney on the pylorus, and, secondly, from the influence on the nervous system.

Disease of the Liver. The intimate relationship between the liver and the stomach is such that when one is the seat of serious functional disturbance the other is likely to be affected, and it is often impossible to determine with absolute certainty which organ is the primary seat of disorder. The abuse of alcohol frequently induces chronic gastritis, just as it causes cirrhosis of the liver. On the other hand, cirrhosis of the liver is frequently the cause of chronic gastritis secondary to the portal congestion.

Diseases of the Nervous System. Attention has been called repeatedly to the relationship existing between disease of the central nervous system and disturbance of the gastric functions. (See Vomiting.) In sclerosis of the posterior columns of the cord this interdependence is more striking than in any other spinal disease. Not only do we have gastralgia and gastric crises; but mild indigestion, with hyperæsthesia and slight gastralgia, may be the first symptom of locomotor ataxia.

Diabetes. Diabetes may continue its course for a long period of time, during which the patient is thought to have stomach trouble, before an examination of the urine reveals the true nature of the case.

Gout and Rheumatism. Opinions differ as to the relationship of gout and rheumatism to gastric disorder. Some writers believe that a specific gouty inflammation of the stomach, due to the uric acid diathesis, is of frequent occurrence, and that one of the prominent manifestations of gout is dyspepsia in all its forms. The French consider gastric disturbances to be frequent expressions of the rheumatic diathesis. The relationship of the two, however, is thus far not fully developed; although it is unusual in these conditions to overlook the presence of either diathesis when symptoms of gastric disturbance occur. It is essential to bear in mind that gastric disturbances are more likely to occur in persons of a rheumatic or gouty diathesis than in healthy individuals, and that their successful management depends upon the recognition of the fundamental diathesis. It is more than probable that gastric disorder, along with defective metabolism, is primary in both affections.

General Examination. The objective examination has thus far been confined to a study of the stomach. The student will infer from the preceding chapters that in order, on the one hand, that the possible cause of the gastric disorder may be determined, or, on the other, the effect of gastric disorder upon the other organs ascertained, the latter must be examined carefully. Moreover, valuable data in the recognition of gastric affections and the diagnosis of the various forms are secured by such examination. The general appearance of the patient, the state of nutri-

tion, and the degree of strength furnish suggestive facts in the diagnosis. As well said by Stockton: "The preoccupied and dejected manner observed in those suffering from continued gastric flatulence; the restless, discomposed behavior, the stooped posture and half-surprised expression often seen in the victims of gastralgia; the emaciated, weak, and cachectic appearance frequently accompanying chronic food stagnation, are good examples of the value of the general appearance in the diagnosis."

It must be remembered that any local source of irritation distant from the stomach, as the eyes, the nose and pharynx, the uterus and ovaries, and the rectum, may be the primary cause of gastric disorder. The study of the hepatic and intestinal functions assists in the diagnosis. Examination of the urine and the blood may enable us to determine the nature of a gastric morbid process. Even the study of the skin is of importance. "A sallow, earthy-colored skin, showing improper secretion; a dry, harsh skin, with too rapid loss of epithelium, showing poor nutrition; a skin showing œdema, poor capillary circulation, lividity, or acne; certain forms of eczema, excess of pigment, or syphilides may afford important information as to the digestion, inasmuch as some of these may be the results and others accompaniments of gastric disturbance." (Stockton.)

The Blood. Examination of the blood enables us to determine the degree of the anæmia which may be the cause of digestive failure. The examination must be exhaustive. If a leucocytosis is present and there is no complicating condition, the gastric neuroses may be excluded. In carcinoma there is not only a severe secondary anæmia, but also poikilocytosis and a multinuclear leucocytosis. Such changes are without doubt the result of interference with the digestion because of motor inactivity. Post-digestive leucocytosis usually does not occur in carcinoma, and its absence is contributory evidence that carcinoma is present. It is, however, a very uncertain sign, as shown by Osler and McCrae and others.

The Urine. No study of a gastric disorder is complete without an exhaustive examination of the urine. For diagnostic, but chiefly for therapeutic purposes, the presence of renal insufficiency, hyperlithuria, indicanuria, glycosuria, peptonuria, and albuminuria must be ascertained.

The Reaction. The reaction of the urine is modified by the state of the stomach. In health the urine is alkaline after a full meal of ordinary character, but when HCl is absent from the gastric contents, this normal alkalinity does not occur. Alkalinity is rarely seen in gastric carcinoma.

The Chlorides. The chlorides are lessened when a small amount of food is taken; a similar cause diminishes the quantity of urea. Both are decreased in carcinoma and in some benign diseases of the stomach. But the chlorides are diminished in carcinoma without a proportionate lessening of the urea. It is this disproportion which, as pointed out by Nothnagel, is of diagnostic value in carcinoma ventriculi.

DISEASES OF THE STOMACH CHARACTERIZED BY FEVER, WITH PAIN AND VOMITING.**Acute Gastritis.**

The simple variety of acute gastritis varies, according to the cause, from a slight attack of vomiting after indiscretion in diet with ordinary symptoms of indigestion to the more severe forms ushered in by chill and attended with fever.

In the mild forms there is a sense of fulness and discomfort in the epigastrium, attended with nausea. The appetite is lost, there may be disgust for food, and the flow of saliva is increased. There is excessive acidity. On examination the epigastrium is found to be tender. The onset of the attack is attended with giddiness, flashes of light before the eyes, frontal headache, and some prostration. The pulse is increased in frequency. When the nausea is most pronounced, the face is pale and the extremities cold; vomiting then occurs, the ejected matter consisting of ingesta only slightly changed, with mucus and watery fluid. The taste is very bitter, and the vomitus is often colored green from bile-pigment. Another attack of vomiting may be sufficient to give relief, or it may be repeated for twenty-four to forty-eight hours every hour or two. After the stomach is relieved of food, mucus and bile alone are vomited.

Examination of Stomach Contents. In ordinary cases the reaction of the vomited matter is neutral or faintly acid. No free hydrochloric acid is present, but later fatty acids are found; pepsin is diminished. In some cases the quantity of HCl may be increased as the result of a less severe irritation.

Twelve or twenty-four hours after the gastric symptoms intestinal symptoms may arise. Borborygmi and colicky pains are complained of, followed by diarrhoea, with some tenesmus. Herpes labialis may occur, and some writers speak of a peculiar odor being exhaled from the skin. The more severe cases are ushered in with chill followed by fever. The local symptoms are much aggravated. The tongue is furred, and the breath foul. The vomiting is frequent and severe. The skin is livid and the pulse becomes rapid.

Diagnosis. Acute cases attended by fever may be mistaken for meningitis, peritonitis, or hepatitis. Identical gastric symptoms may usher in an attack of pneumonia. The possibilities of a mistake are to be borne in mind, and in all cases of vomiting with fever due regard must be paid to the possibility of the gastritis being merely symptomatic. The same group of symptoms that belongs to gastritis also accompanies the exanthematous diseases, as well as diphtheria, dysentery, pyæmia, and puerperal fever. The gastric symptoms may be of reflex origin, or due to the action of fever, poison, or ptomaines on the stomach. Ewald uses the term "sympathetic gastritis" when the symptoms are the same as in the simple variety, masked, however, by the primary disease. Sometimes, however, as in the eruptive fevers, attention is directed to the state of the stomach, to the exclusion of other conditions, until, to the surprise of the student, the appearance of an eruption or inflammation reveals the true nature of the case.

In cases of gastritis, therefore, endeavor to find a local cause for the symptoms. If there is no history of indiscretion in diet, of exposure, of exhaustion, or mental shock that might have arrested digestion, then inquire for a history of exposure to contagious diseases and look for the earlier evidences of exanthemata. If the result of the examination is still unsatisfactory, examine the condition of each individual organ, particularly bearing in mind meningitis, pneumonia, peritonitis, nephritis, and the general infections.

Mycotic and *pseudomembranous gastritis* may occur secondarily to typhoid fever, pneumonia, pyæmia, and smallpox. In very rare instances actual diphtheria of the stomach has been observed. The fermentation incident to the presence of yeast-fungi and sarcinæ in dilatation causes irritation, and these organisms may perhaps irritate the viscus directly. The mucous membrane may be covered with patches in areas or throughout its whole extent. Rarely tuberculous inflammation with ulceration takes place. Many other micro-organisms are frequently present, and though they are chiefly non-pathogenic they may contribute to the trouble and often cause fermentation and consequent irritation. Klebs found *Bacillus gastricus* with numerous spores in the tubules as the cause of a gastritis.

The mucous membrane itself usually escapes infection by micro-organisms because of the character of its secretion. The acid gastric juice is antagonistic to micro-organisms and causes their death. Tuberculosis, for instance, rarely attacks the stomach for this reason.

Phlegmonous Gastritis.

This is a very rare affection, in which the inflammation is seated in the submucosa and leads to perforation. The onset is sudden. The chief local symptom is intense pain in the epigastrium, with a burning sensation. The anorexia is absolute, the tongue is dry, and the acidity is increased. The fever is high and accompanied by delirium and chills. The pulse is small, rapid, and irregular. The matters vomited first consist of mucus, then pus. The patient is extremely restless and anxious, even delirious, and early passes into coma. Death takes place from collapse. It is impossible to make an absolute diagnosis, as local peritonitis and abscess of the liver are characterized by the same symptoms. In *abscess* a tumor may form in the epigastrium. The disease may be idiopathic, but frequently occurs in septicæmia, and follows trauma. The subjects are often alcoholics.

Toxic Gastritis.

This form of gastritis is allied to the former in the severity of the general symptoms. It is the result of the swallowing of irritating poisons, of which phosphorus, arsenic, bichloride of mercury, and caustic acids and alkalies are the most common. It is attended by inflammation of the mouth, œsophagus, and stomach. The flow of saliva is excessive, the patients are unable to swallow and constantly vomit blood, which is often mixed with shreds of mucous membrane. The patient is restless,

and may have convulsions; collapse soon develops. In mild cases, in which the local effects of the corrosive substances have been mitigated by proper antidotes, sloughing takes place and ulcers remain on the mucous membrane, which, after healing, result in deformity or stenosis of the œsophagus.

Some cases are attended by other symptoms peculiar to the special poison. Thus in arsenic-poisoning there are choleraic symptoms; in phosphorus-poisoning the symptoms come on late after the ingestion of the drug, and are attended by jaundice and symptoms of acute yellow atrophy.

DISEASES OF THE STOMACH CHARACTERIZED BY INDIGESTION.

Functional Disorders of the Stomach.

The Neuroses. Functional disturbances of the stomach are due to impairment of the motor power of the stomach, and to impairment of the secretory and sensory functions. The following table of Ewald is a classification of the various neuroses midway between the symptomatic and the ætiologic:

THE NEUROSES OF THE STOMACH.

1. CONDITIONS OF IRRITATION.

<i>a. Sensory.</i>	<i>b. Secretory.</i>	<i>c. Motor.</i>
Hyperæsthesia.	Hyperacidity.	Eructation.
Nausea.	(Hyperchlorhydria.)	Pyrosis.
Hyperorexia.	Hypersecretion.	Vomiting.
Anorexia <i>ex</i> hyperæsthesia.		Colic.
Parorexia.		Tormina ventriculi.
Gastralgia.		

2. CONDITIONS OF DEPRESSION.

Polyphagia.	Anacidity.	Atony.
Anæsthesia.		Insufficiency of the pylorus and cardia.

3. MIXED FORMS.

Gastro-intestinal neurasthenia (dyspepsia nervosa).

4. REFLEXES FROM OTHER ORGANS UPON THE GASTRIC NERVES.

Reflexes from the brain, eyes, spinal cord, kidneys, liver, sexual organs, and intestines manifest themselves in the forms mentioned in 1 and 2.

It must not be supposed that each of the above-named symptoms occurs in an individual, or that functional disturbances may be limited to alterations in the sensory and secretory, or in the motor apparatus respectively. As Ewald remarks, they occur not as distinct independent diseases, but usually in groups, "either appearing simultaneously or closely following one another during the course of the malady, passing before us like an ever-changing scene." They may arise directly from disease of the stomach, or reflexly from disease of other organs, the brain, the spinal cord, uterus, kidneys, liver, eyes, or nose.

Ætiology. Gastric neuroses are of most frequent occurrence in women, especially during the years of sexual activity. The accidents of child-

birth are predisposing factors. In both sexes they are of most frequent occurrence after the age of twenty because individuals are subjected to causes that lead to neuroses at this period of life. The gastric neuroses occur in all classes. They are more likely to occur in those who are poorly nourished or anæmic, although persons who are distinctly robust may also suffer. While more common among the residents of cities, they may occur in farmers and others accustomed to an open-air life. Although we are oftenest called upon to treat them among the better classes, a large number of cases are also seen among the poor. To analyze more closely the predisposing causes, we have to study individually all conditions and circumstances in life that lead to wear and tear, as in business or social affairs. The causes which Beard and others have forcibly pointed out as factors in the production of neurasthenia are especially prevalent in this country.

In men, excessive devotion to business or dissipation; in women, excesses in social life or the restraint of home cares, with, unhappily too often, the irritation of marital relations, are the predisposing factors which lead to the development of this class of cases. Often patients in the large cities are subject to the neuroses in the spring after the dissipations of the winter. Behind this excess there is, no doubt, in the majority of cases, a nervous temperament that is responsible for the bringing out of the symptoms, particularly if the patients live in an unhygienic way in regard to exercise, ventilation, drainage, and diet.

Symptoms. With the gastric neuroses other symptoms of *neurasthenia* are present, which may lead the patient to seek advice: headaches of various kinds, changes in the mental condition, vertigo, insomnia, neuralgias, and all forms of paræsthesia. Intimately connected with the neurasthenic state is that of hysteria, and therefore in gastric neuroses *hysterical manifestations* are most common. It may be impossible completely to define the border-line between neurasthenia and hysteria, and the gastric symptoms of the former are the gastric symptoms of the latter. While, therefore, general neurasthenic symptoms are prominent, in order to reach a diagnosis upon which proper lines of treatment can be based, the condition of the individual must be viewed as a whole, and no one symptom or group of symptoms exaggerated in our minds.

Varieties. Ewald has divided the neuroses into those which arise from (a) *irritation*, those which arise from (b) *depression*, and (c) those in which both are combined—*mixed neuroses*.

a 1. **Sensory Neuroses of Irritation.** **HYPERÆSTHESIA.** The first result of irritation is *hyperæsthesia* of the stomach, indicated by a feeling of *fulness* and *tension*, with *nausea*. The sensation is often somewhat like the normal sense of hunger, and is also seen in chronic gastritis, as well as in hysteria, meningeal irritation, cerebral tumors, and other diseases of the nervous system. The increased irritability is such that the mildest irritant excites discomfort or a painful sensation. There is a continuous sensation of heat or cold, of gnawing, or pulling, or burning in the organ. The local sensation reflexly influences the physical life of the patient, so that *hypochondriasis* in some form attends it. The sensations may be relieved by food, to become worse when the stomach is empty, although

in the larger number of cases the trouble is aggravated during digestion. The sensations are likely to be aggravated by fasting a longer period than usual, or by restriction of the diet. Excesses may aggravate them, and, on the other hand, they may follow debilitating states. Some foods, such as shell-fish, crabs and lobsters, or oysters and strawberries, are likely to increase the peculiar sensations in the epigastrium, exciting mild depression, or burning, or even nausea. The excitation from these foods is usually due to idiosyncrasies which also are responsible for pruritus, erythema, and urticaria with headache and some elevation of temperature.

PERVERSIONS OF THE SENSE OF HUNGER. *Hyperorexia.* When hunger is exaggerated, it is known as boulimia, or hyperorexia. It may be temporary or permanent. When permanent, it is obstinate, weakening, and exceedingly unpleasant. It may occur alone or be a symptom of various diseases of the nervous system, as manifest disease of the brain, neurasthenia, hysteria, and psychoses. It complicates such disorders as diabetes, and may be of temporary duration in convalescence from acute disease. The disorder may accompany migraine, hypochondriasis, and exophthalmic goitre. Analogous to it is perversion of the appetite, as seen in pregnancy, during childhood, and in mental disorders.

Anorexia. Loss of appetite, or repugnance to food. In the first instance, there is simply loss of appetite; in the second, there is repugnance to food or nausea at the sight of it. Loss of appetite accompanies all forms of dyspepsia. In the gastric neuroses anorexia occurs spontaneously, or is due to hyperæsthesia of the stomach, and may therefore arise from central or peripheral conditions of irritation. It is commonly seen following central nerve perturbation. The patient is hungry and sits down to the meal fully prepared to satisfy himself. The first mouthful is at once followed by anorexia, which may almost amount to nausea. On account of these symptoms the patient eats less and less of solid food, which soon results in disturbance of nutrition affecting the centres. Conversely, profound mental disturbance may be an exciting cause, so that after the death of a friend or shock of any kind the patient is unable to take food. Loss of appetite may be the only manifestation of the gastric neurosis, but owing to the serious interference with nutrition other local or general symptoms soon result. Fenwick points out that its relationship to emaciation and enfeeblement is such that grave organic diseases may be simulated. Thus the patient's condition may be mistaken for phthisis, and a general examination alone be sufficient to clear up the mistake.

GASTRALGIA. *Pain* in the stomach occurs in organic disease, as in ulcer or cancer, or forms of gastritis; but it also attends a gastric neurosis, and may be the only symptom of this neurasthenic state. Such pain is functional, and is found in anæmic, neurotic women, although it may occur in all classes. The pain is sudden, referred to the epigastrium, usually without regularity, though at times distinctly periodic. There may not be any definite relationship between the attack of pain and the taking of food, although the pain is most apt to occur when the stomach is empty. Some kinds of food may aggravate it, though, in general,

eating relieves the pain. If the epigastrium is examined, it will be found free from tenderness—indeed, pressure with the palm of the hand may give relief. The pain is of an agonizing character, sometimes sharply localized, or again diffuse. It may even resemble the girdle-sensation. On account of the severity of the pain the patient may double himself up to relax the abdominal muscles. The breathing is short, and speaking is done in a whisper. The attack is attended by more or less collapse, and the patient may complain of a sensation of impending death. There is pallor of the face, which is distorted with pain, and the brow is covered with perspiration. The pain may radiate along the spinal nerves in close proximity to the stomach, and there is often vigorous pulsation of the abdominal aorta. The attack may last but a few minutes or continue for hours. It sometimes terminates suddenly with vomiting, or is relieved as soon as food is taken. After the attack the patient is exhausted and relaxed, and passes an abundance of urine of low specific gravity.

The gastralgias that are due to disease of the central nervous system are often most puzzling. Rosenthal has written exhaustively on this subject. Types of gastralgia of this character are seen in the *gastric crises* of tabes, first described by Charcot. Recent observers have found that it is due to sclerotic degeneration of the vagus nucleus. The patient is suddenly seized with severe pains beginning in the groin and ascending along both sides of the abdomen to the epigastrium, where they remain localized. Pain in the shoulders occurs at the same time. The pains are suggestive of locomotor ataxia in their lightning-like rapidity. With the pain the heart's action is increased in rapidity and force. There is no rise in temperature. At the same time there is uninterrupted and painful vomiting, attended by nausea and vertigo. The gastric pain may continue uninterruptedly for two or three days. The frequency of the attacks is variable; they may recur at long periods, or as frequently as once a month or once a week. The symptom belongs to the pre-ataxic period, so called, but is almost sure to continue throughout the whole course of the disease. The nature of the stomach contents sometimes bears no relation to the pain, but HCl is often in excess. Another special characteristic is the sudden relief without apparent cause.

Neurasthenic Gastralgia. Neurasthenic gastralgia occurs in patients who are suffering from neurasthenia, and is divided by Rosenthal into two forms, the one irritative, the other depressant; these are connected by transitional forms. The early symptoms of neurasthenia (*q. v.*), particularly of the irritative form, with the presence of sensitive points in the nape of the neck and between the scapulæ or often lower down on the vertebræ, with neuralgias and paræsthesias in the upper and lower extremities, are attended by periodical gastralgia. The gastralgia is characterized by a boring sensation which, during the attack, radiates over the lower ribs to the median line. It is accompanied by vasomotor symptoms and symptoms of cerebral anæmia. In the *depressant* form the patient complains of weight and fulness, or a dragging sensation after eating, which is constant instead of paroxysmal. The neuralgic pains are not so marked, motor exhaustion is not so prominent, and the pain

in the back is not so intense as in other varieties. In both instances on deep pressure over the region of the nerve-plexuses that accompany the bloodvessels in the abdomen there is sharp pain radiating to the epigastrium. Burkart considers these painful points to be present in all cases, while Richter believes that pressure over the stomach and abdomen is not painful. With such pain there is usually increased pulsation of the abdominal aorta, particularly during the paroxysm. In neurasthenic gastralgias there are increased sensitiveness to the electrical current and increased irritability of the sensory nerves of the trunk, sometimes extending to the limbs.

Neurasthenic gastralgia must be distinguished from the gastralgia of organic disease and the gastralgia of hysteria. The gastralgia of organic disease is recognized by a study of the secretion when fasting. In organic disease there is retarded digestion, or the signs of ulcer are present; in gastric neuroses digestion is completed in the normal limit of time—seven hours—and there is often no disturbance of the chemical condition of the gastric secretions, though hyperchlorhydria of purely nervous origin undoubtedly occurs. Hysterical gastralgias are recognized by the presence of the usual symptoms of hysteria, in which the psychical factors occupy a prominent place, associated with convulsions, paralyses, pupillary inequalities, hemi-anæsthesia, and electrical insensibility. Most characteristic, however, is the alternation of hysterical gastralgias with neuralgia, or neuroses in other organs.

a 2. **Secretory Neuroses of Irritation.** HYPERCHLORHYDRIA, or hyperacidity due to excess of HCl, is an extremely common condition. It is most frequently due to organic disease of the stomach, such as benign stenosis of the pylorus or ulcer; but it may be a pure neurosis accompanying neurasthenia, hypochondriasis, melancholia, or hysteria; or may be due to the reflex irritation of gallstones, renal calculus, and similar conditions. The diagnosis between organic disease and neurosis is often very difficult. The symptoms of hyperchlorhydria are heart-burn and acid eructations, with burning or boring pains in the epigastrium, which are usually worse when the stomach is empty and relieved by taking albuminous food, but often rather aggravated by starches. Physical examination usually shows epigastric tenderness, which may be severe, and often slight or moderate enlargement of the stomach. The contents after a test-meal usually have a high acidity (60 to 120 or more), starch digestion is very imperfect, and there is an abnormally large amount of material. Many writers have noted that symptoms of hyperchlorhydria may be present when the HCl is not in excess. Strauss and his students believe that the excessive secretion in these cases is shown by a lively starch reaction and a low specific gravity (below 1010). The *diagnosis* of the nervous form chiefly concerns the distinction from acid gastritis, ulcer, and motor insufficiency of the stomach. When there is much mucus in the stomach contents, acid gastritis is easily diagnosed and may be considered to be present when there are evident causes of gastritis. In many cases, however, it is practically impossible to say whether it is a gastritis or a neurosis, and the diagnosis must be based upon the general condition of the patient rather than upon the

local signs. Ulcer and motor insufficiency are distinguished by the signs mentioned in considering those conditions. In some cases ulcer can be diagnosed only by observing a rapid recovery under treatment. This is very uncommon in nervous hyperchlorhydria, but it is not an absolutely distinctive feature.

GASTROXYNSIS is a gastric neurosis in which, after mental over-exertion or profound emotional disturbance, there is severe headache with sudden vomiting of acid fluid, continuing for a considerable time. It is closely allied to migraine, and is a form of periodical hypersecretion.

HYPERSECRETION is probably an accompaniment of practically all instances of hyperchlorhydria, whether of nervous or of organic origin. In ordinary cases it is recognized largely by the fact that the contents removed after a test-meal are both very acid and of large amount; often, even more than was introduced is removed. The symptoms in such cases are produced conjointly by the increase in acidity and the excess in the quantity of gastric juice. There are, however, cases in which the hypersecretion is evidently the chief factor. These were first described by Reichmann under the name of gastrosuccorrhœa, and belong under two classes: the periodical and the chronic. Periodical hypersecretion is characterized by the occurrence of general physical prostration, with more or less epigastric pain and tenderness and vomiting. The vomitus is at first food and gastric juice and perhaps mucus; later it consists of large amounts of a highly acid gastric juice, clear or mixed with bile. The attacks last from a day to two weeks, and often produce profound prostration. They really constitute a form of cyclical vomiting or gastric crisis, the distinctive feature being the high acidity of the vomitus and the great quantity of secretion produced by the stomach.

The chronic form presents usually the symptoms of hyperchlorhydria with the added feature that the patients often vomit considerable amounts of clear, highly acid gastric juice when fasting, and the stomach, when it should be empty, as in the early morning, shows the presence of from 100 to 300 c.c. of the same excessively acid gastric juice, free from food remnants. The affection is chronic and persistently present. The periodic form is apparently usually a neurosis, and the chronic form is thought to be so in some instances, but organic disease must be carefully looked for in these cases. In most instances it is due to moderate pyloric stenosis, to pyloric spasm, or to motor weakness. Doyen in particular has insisted upon the importance of erosions and ulcers in producing pyloric spasm and gastrosuccorrhœa.

a **3. Motor Neuroses of Irritation.** ERUCTATIONS. Eructations and belching are phenomena of the gastric neuroses of motor origin. They usually occur in hysterical subjects rather than in neurasthenics. In the latter they are associated with other sensations, particularly oppression and tension in the epigastrium. In hysteria they occur alone. There is increase in the contractility of the stomach, the pyloric sphincter contracts powerfully, and the stomach is distended; gas is expelled at the cardiac end of the stomach. Eructations may be due to paralysis of the cardiac end of the stomach rather than to contraction of the pyloric end. As a rule they occur involuntarily, and must not be confounded with the

pseudohysterical vomiting described by Bristowe, in which the gas is raised from the œsophagus by contraction of the muscles of the neck. Hysterical eructation is very frequently of œsophageal origin. The belching is loud and may occur in paroxysms. The gas is odorless, and hence is distinguished from the gas of dyspepsia and fermentation; it is in all probability the result of the swallowing of air.

PYROSIS. Pyrosis, or heartburn, is the raising of sour masses from the stomach. The stomach contents are not necessarily hyperacid. If the material is acid, like normal gastric juice, or hyperacid, the regurgitation causes severe acrid and burning sensations. The neurosis is probably due to heightened contractility of the muscular coat of the stomach with pyloric contraction, which overcomes the weaker cardia.

PNEUMATOSIS. Excess of gas in the stomach. When the stomach is over-distended, the diaphragm is pushed up and presses against the heart. The patients are accordingly seized with severe dyspnœa; at first inspiration is difficult, and finally both inspiration and expiration are impeded. Palpitation of the heart and pulsation of the peripheral arteries take place. There are fulness of the head and a sensation of impending death; the patient may even become unconscious. Relief can only be afforded by belching, when the attack rapidly subsides. Introducing a stomach-tube gives immediate relief.

NERVOUS VOMITING. (See Subjective Symptoms and Gastroxynsis.)

TORMINA VENTRICULI. Peristaltic unrest. Characterized by borborygmi and gurgling, beginning immediately after eating; the sounds are heard at a considerable distance, and are a source of great annoyance. Peristaltic unrest is a common symptom of the gastric neuroses.

RUMINATION. *Merycismus.* Rumination is a rare condition in which the patients regurgitate and chew the cud like ruminants.

b 1. **Secretory Neuroses of Depression.** **ANACIDITY.** Anacidity of the gastric juice as a neurosis is found in hysterical persons and in neurasthenics. (See chemical examination—Absence of Hydrochloric Acid.)

b 2. **Sensory Neuroses of Depression.** **ANÆSTHESIA.** In conditions of depression *polyphagia* or *acoria*, the want of a feeling of satiation, occurs; if gluttony is excluded, it is a morbid condition of extreme rarity.

b 3. **Motor Neuroses of Depression.** **ATONY, or ATONIC DYSPEPSIA,** accompanies gastritis, and occurs also as a primary neurosis. The nerve-centres regulating peristalsis are disordered. The primary disturbance of innervation may be local or central. The movement of the chyme is tardy or insufficient. Atony should be applied to disturbance of the motor function only, or, as Rosenbach states it, to insufficiency of the stomach. The symptoms develop gradually. At first oppression during digestion occurs, with swelling and fulness of the stomach.

There is mental and physical torpor during the time of the digestive act. The symptoms become aggravated, and eructations occur, vomiting begins, and gradually the fermentative symptoms become most pronounced. At this period the condition is best described by the term putrid, or fermentative dyspepsia. By the usual tests the motor power of the stomach is found to be diminished. The secretions are often reduced, though they may frequently be excessive.

RELAXATIONS AT ORIFICES. *Relaxation of the Cardiac and Pyloric Ends of the Stomach from Conditions Resembling Paralysis.* When the cardiac end is relaxed, eructations and regurgitations occur. If large quantities of the material from the stomach are regurgitated and expectorated, the condition is pathological, and may lead to serious disturbance of nutrition. The condition may exist for years without producing bad results. It must not be confounded with the regurgitation from diverticula of the œsophagus.

The relaxation is often the result of repeated voluntary regurgitation in hypochondriasis. Relaxation of the pylorus is said to cause rapid discharge of food into the intestine and a consequent lenteric diarrhœa. The diagnosis is always questionable.

c **Mixed Neuroses.** NERVOUS DYSPEPSIA. According to Ewald, this is the true gastric neurasthenia, which combines all forms of gastric neuroses. The clinical picture is made up of a combination of various neurosal symptoms. Leube considers nervous dyspepsia a group of symptoms of a cerebral nature due to abnormal irritability of the sensory nerves of the stomach during the normal digestive processes, and consisting of hyperæsthesia and nausea, hyperorexia, anorexia, parorexia, and gastralgia. The secretory power of the stomach varies greatly, being sometimes normal, sometimes increased, and sometimes subnormal or absent. Although the anatomical or physiological explanation of the condition is difficult, the clinical symptoms are those of irritation or paralysis, the manifestations of which are intermingled, sometimes one and sometimes the other being most prominent. (See table, page 1018.)

The one characteristic feature is that the symptoms are mild. Nervous vomiting and boulimia do not occur with severe forms of gastralgia. Symptoms of intestinal indigestion are usually associated in a mild degree. Constipation is of the most common occurrence, although in some cases there is diarrhœa. In other cases the intestinal indigestion is much aggravated, with mild gastric disturbances and anorexia, distaste for food, furred tongue and mild nausea, constipation, and colicky pain, either diffuse or in separate painful spots. The abdomen is distended and tympanitic, sometimes to a marked degree. It is called *flatulent dyspepsia*. Along with the gastric and intestinal symptoms, the general nervous symptoms to which the term neurasthenia is applied are present. These nervous manifestations sometimes precede the local gastric symptoms, but as the latter develop the former become more aggravated. The dyspeptic conditions, as Ewald puts it, rest on a neurotic basis, or are such as may occur in the form of reflex neuroses in chlorosis, menstrual disorders, uterine and ovarian disease, and after intense physical or psychical excitement. It is impossible to say to what extent the symptoms depend upon anatomical changes and alterations of secretion. Cases have been described in which severe digestive disturbances were present for as long as ten years and yet post-mortem examination showed no anatomical changes. The general teaching, however, is that in most cases, even though alterations of structure or function may be absent in the beginning, they appear later as a result of the neurasthenic disturbance.

Diagnosis. There are no characteristic symptoms, and the student

must bear in mind that it may be necessary to make several examinations and listen to the story of the subjective symptoms frequently before a conclusion can be arrived at. This is all the more necessary because of the frequency with which organic lesions and neurasthenic conditions are present at the same time. The course of the disease must be observed for a long time, all possible causal factors investigated, and all the general signs of neurasthenia carefully considered. In addition, it may be necessary to use therapeutic tests. If the possible organic diseases are not relieved by such measures, there must be a deeper basis for the gastric symptoms. Just as in neurasthenia and neurasthenic states elsewhere, the peculiarities, idiosyncrasies, and all the associations in the life of the individual must be considered in connection with the general and local symptoms of the neurasthenic state. Great stress must be placed upon the study of individual symptoms, their mutual relationship, and their changeable occurrence. In gastric neurasthenia gastralgia is more diffuse than the pain of ulcer or cancer of the stomach. It is not so much dependent upon ingestion of food as either of the other forms, particularly that which accompanies ulceration. In gastric neurasthenia vomiting is rare; when it occurs, the material is composed of mucus mixed with bile and food in various stages of digestion, and is never bloody, nor does it contain decomposed masses. Hysterical vomiting occurs with ease and regularity as compared with that of neurasthenia, in which the vomitus is bitter from the presence of peptones. The stools in gastric neurasthenia are changeable in character, and do not contain undigested remnants of food, or mucus, or blood, while the shape of the feces is variable.

Differential Diagnosis. Neoplasms, ulcers, strictures, and dilatation are distinguished by physical signs or characteristic symptoms. In gastric neurasthenia the stomach should be empty seven hours after taking a meal. The results of the chemical examination are not sufficiently definite for diagnostic purposes; for at times the same chemical changes are present as in ulcer, carcinoma, and chronic catarrh. The diagnosis, as previously intimated, is based chiefly on prolonged observation and a carefully taken history, and on the general condition of the patient. The cases must not be mistaken for costal neuralgia, although it is not often that one is led astray. Reflex gastric neuroses as indigestion, gastralgia, or vomiting are seen. The types are interchangeable, although vomiting occurs in the more acute reflexes, indigestion in the more chronic. The cerebral disorders that give rise to vomiting are meningitis, abscess, and tumor. The vomiting may be transitory or persistent, and there is usually hypersecretion of the gastric juice; the vomiting may usher in the disease or develop during its course. When vomiting is of long standing, the possibility of a reflex origin should always be borne in mind. (See Vomiting.)

Gastralgia is sometimes a reflex symptom of a lesion in the cervical and dorsal portions of the cord, not only in tabes dorsalis, but also in disseminated sclerosis; the attack of pain and vomiting is known as a *gastric crisis*.

Chronic dyspepsia is frequently the expression of reflex irritation in

diseases of the sexual organs, as amenorrhœa and dysmenorrhœa during the climacteric period, and in chronic inflammations of the uterus. Malpositions and tumors, pelvic exudates with traction, ulcers, and ovarian tumors, are often attended with the so-called *dyspepsia uterina* of Kisch.

Chronic Gastritis.

Causes. 1. Previous attacks of acute gastritis.

2. The local irritation of badly cooked or poorly masticated food, and of alcoholic and other beverages.

3. The local irritation of urea in chronic Bright's disease, and of products of putrefaction in constipation.

4. Chronic gastritis is of frequent occurrence in anæmia and in venous congestion from any cause, but particularly from disease of the heart or diseases that interfere with the portal circulation. It occurs secondarily to diabetes, gout, rheumatism, nephritis, and tuberculosis.

5. It is a constant attendant upon local disease of the stomach, as cancer, dilatation, and ulcer, and of local disturbance of the circulation.

6. Neurasthenia is undoubtedly a prolific cause of chronic gastritis. It may produce only atony or functional disturbance at first; but these ultimately lead to gastritis.

The symptoms are those of *chronic indigestion*. There is a dry, pasty, or salty taste in the mouth, especially in the morning. The tongue is coated over its entire surface, or has red patches at the base; its papillæ are always swollen and its edges marked by the teeth. Aphthæ recur frequently. The lips are dry and often chapped. The appetite is diminished or capricious. Although there is no great thirst, the patients crave fluids with their meals, and acid drinks are grateful. After eating there is a feeling of oppression and distention in the epigastrium, frequently followed by belching. The gaseous eructations may be odorless or foul; rancid regurgitation with pyrosis is frequent. The acidity is due to fatty acids and not to hydrochloric acid, as in hypersecretion. Vomiting is frequently present, but occurs irregularly, and is usually preceded by nausea. The most characteristic form is that in which mucus is vomited in the morning on rising. Constipation usually exists; it may alternate with diarrhœa. There are flatulence and rumbling in the intestines.

General Symptoms. The nervous symptoms are the most pronounced. The mental activity is diminished, there is a feeling of languor or torpor, especially after eating. Headache is frequent after eating, and the patient may become morose and hypochondriacal. Attacks of vertigo are common. Itching of the skin and coldness of the extremities are not rare. Sleep is deeper and longer than is natural, but is disturbed by dreams, and is not refreshing. Yawning is frequent. Pharyngitis is usually present, with hacking cough and expectoration, or the hawking of mucus. The pulse may be weak and irregular, and at times there is an evening rise of temperature. The urine is scanty, high-colored, and usually loaded with urates.

Three chief forms are seen: (1) subacid gastritis; (2) acid gastritis; (3) atrophic gastritis. The last is likely to result from long-standing

cases of the other forms and constitutes the organic form of the condition called by Einhorn *achylia gastrica*. It is important to distinguish this condition from the neurotic form of achylia. In both there is entire absence of acidity and of digestive ferments, and the test-meal is returned unchanged in appearance except for some maceration. The general health is not infrequently unaffected in both varieties; but this is more commonly the case in the neurotic form, while the organic cases are characterized by absence of neurasthenic characteristics, a prolonged history of gastric disturbance and often severe intestinal symptoms, chiefly diarrhœa. Severe anæmia is often present and the general depression of health may be extreme. The diagnosis from cancer may be very difficult in these cases. It rests chiefly upon the absence of lactic acid, of Oppler-Boas bacilli, and of pus from the stomach contents, the lack of evidence of tumor or of pyloric obstruction, and the absence of leucocytosis and actual cachexia.

In *subacid gastritis* the stomach contents contain little or no free HCl, but some of the combined acid, the test-meal is usually poorly digested, and volatile acids are present. In one form called "mucous gastritis" large amounts of mucus are found in the contents after a test-meal. Acid gastritis is a form that is not always recognized, but which certainly exists. It is characterized by the usual symptoms of gastritis with those of hyperchlorhydria superadded, and the contents after a meal are found to be excessively acid. The frequent presence of excessive amounts of mucus, the history of a cause, and the general character of the patient stamp the affection as a gastritis and not a neurosis.

Diagnosis. The diagnostic features of chronic gastritis are: first, long duration; second, persistence of local symptoms; third, recurrence of local symptoms after food, the symptoms being aggravated by stimulants or stimulating food; fourth, moderate pain; fifth, absence of cachexia; sixth, absence of tumor; seventh, flatulence. Hemorrhage is rare and slight when it occurs; there may or may not be vomiting; while the quantity of hydrochloric acid is variable. Finally the cause is usually definite.

Dilatation of the Stomach (Gastrectasia). (See Plate XL., Fig. 1.)

Dilatation of the stomach is caused by obstruction at the pyloric orifice, either from cancer, the cicatrix of an ulcer, or fibrous stricture. In a mild form, so-called "atonic dilatation," it certainly follows the relaxation and degeneration of the walls of the stomach that occur in chronic gastritis and in the conditions causing chronic gastritis. It may attend paralysis of the stomach. Excessive eating and drinking are the only probable causes independent of organic disease. Hence we have (1) obstructive and (2) atonic dilatation.

The dilatation may be *acute*. The term *acute paralytic distention* is also applied to this condition. The cases are by no means so rare as was once thought. They follow blows or operations upon the abdomen, or occur as a result of the relaxation and distention of the walls that appear

PLATE XL.

FIG. 1.



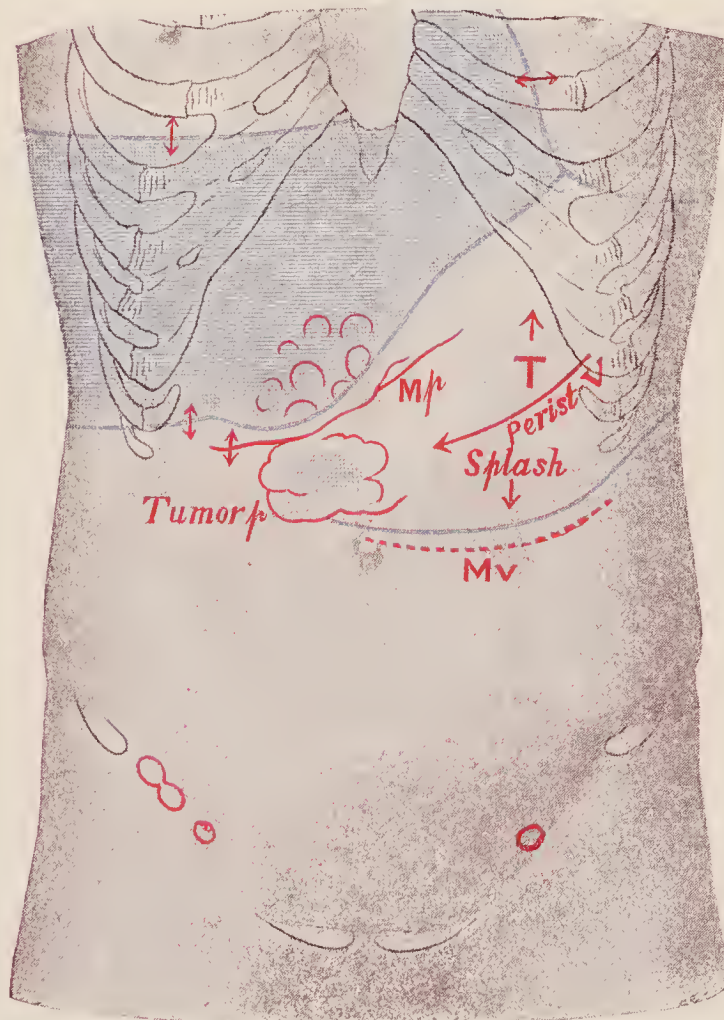
Normal Position and Displacements of the Stomach.

Solid red line—Normal position of distended stomach.

Blue line—Atonic dilatation.

Dotted red line—Gastropptosis.

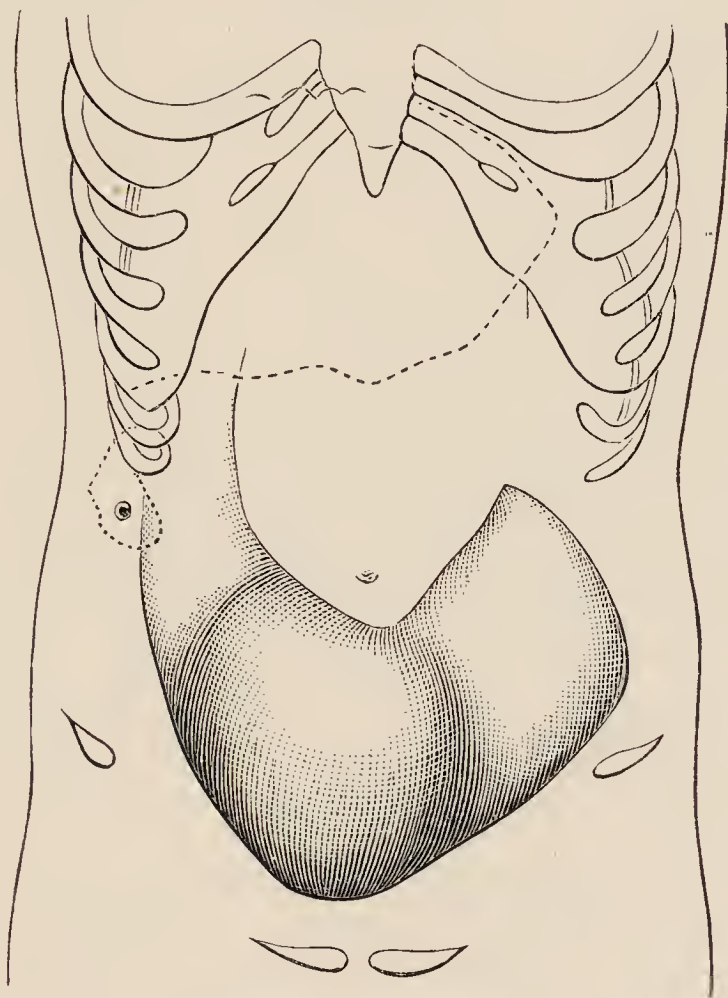
FIG. 2.



Carcinoma of the Stomach with Pyloric Stenosis. Metastases in the Liver.

in acute diseases. There is sudden enlargement of the upper portion of the abdomen, with pressure upon the surrounding structures; the heart is dislocated and its action much interfered with; collapse follows, and may end in death. At first there may be some belching, but the patient is soon unable to remove the gas, and suffers from extreme discomfort, palpitation, and dyspnœa. The vomiting may occur at once or later. It is persistent and excessive. On physical examination the stomach yields the same physical signs as in chronic dilatation.

FIG. 369.



Dilatation of stomach.

Chronic dilatation develops slowly. The *symptoms* are superimposed upon the causal disease. There is marked dyspepsia, with flatulence, pyrosis, and other symptoms of fermentation. The tongue is pale and furred, or red, smooth, and shiny; or it may be soft and flabby. If frequent vomiting has attended the causal disease, it now occurs at longer intervals; the amount is excessive, greater than the normal stomach would hold, and is made up of partially digested and fermented food and large amounts of mucus. The stomach contents contain *sarcinæ*, *torulæ*, and other products of fermentation, and have a disgustingly foul and sickening odor. In atonic dilatation the HCl is usually diminished; in pyloric obstruction it is almost always much increased. In health large amounts of volatile acids are present, but lactic acid, except when introduced with the food, is practically always absent. The stomach contents when allowed to stand separate into three layers—the upper frothy and containing mucus and fermenting food; the middle layer clear and watery; the lower finely divided and consisting of more or less completely digested food. The patient loses flesh and strength; becomes irritable, depressed,

and more or less melancholy; and is subject to vertigo and to attacks of nocturnal asthma. The nervous symptoms of chronic gastritis are also present. Sleeplessness is quite common. In some cases there is excessive thirst because of the small amount of nutriment and fluid absorbed. Cardiac palpitation and irregularity are common, and dyspnoea may occur on account of the distention. Tetany has been observed in cases of dilatation, especially after lavage.

Physical Examination. The diagnosis is not complete without *physical examination*. On *inspection* the abdomen appears large and prominent, and the outline of the stomach can sometimes be seen. Peristaltic movements of the organ are often visible. The movement is from left to right. The heart is lifted upward. On *palpation* the peristalsis can be felt, and with one hand on the stomach, tapping with the other, a *splashing sound* can be elicited. Or the hand may be placed over the stomach while the patient is standing, and the body quickly shaken. On palpation the striking or pushing hand should be compressed over the false ribs. A tumor can sometimes be felt in the region of the pylorus, or below the umbilicus. On *percussion*, when the stomach contains gas, a tympanitic note is heard. After the patient has taken a glass of water, dulness may be detected between gastric and intestinal tympany when the patient stands up. The dull note disappears when he resumes the recumbent posture. While the stomach is empty tympany is not so marked in the upright as in the recumbent posture, because the viscus is dragged back or down. The tympany extends high up in the chest on the left side, so that Traube's half-moon space becomes enlarged, and may extend as high as the fourth interspace on the left side. Cardiac dulness is increased and the apex of the heart is lifted upward and to the left. In the axillary region the tympany may extend as high as the sixth rib, so that unless very careful light percussion is performed, the splenic dulness can not be brought out. The lower limit extends below the transverse umbilical line, and may even extend midway to the pubes. If there is *gastroptosis*, the stomach tympany falls to a lower level. On *auscultation* succussion can easily be elicited. Sometimes the sound is sizzling, as if there was effervescence. Heart-sounds may be transmitted clear and metallic over the tympanitic stomach. With *auscultatory percussion* the border of the stomach can often be defined accurately. Percussion must be commenced far away from the stomach-limit and conducted toward it. (See Examination of the Abdomen.)

The most important point is to determine whether the case is one of atonic or of obstructive dilatation. The chief distinctive features of the former are less marked pain, less violent vomiting, absence of peristaltic waves; the return flow of water in washing the stomach is slow and forceless, the dilatation is usually less marked, and the health is not so profoundly affected. In pyloric obstruction there is often a previous history of ulcer or a small mass may be felt at the pylorus, peristaltic waves are frequently seen, the vomiting is forcible, and the return flow through the tube is very strong. The stomach is usually more markedly dilated, and there is more pronounced retention, while the general health suffers severely, the skin becomes dry and wrinkled, the patient loses

flesh, has a good deal of pain, usually most pronounced at night, and falls into a condition of inanition.

Stenosis of the Pylorus.

Usually, obstruction is caused by malignant disease. Hypertrophic stenosis occurs in rare instances and leads to dilatation, as indicated above. The condition may be congenital or acquired.

Acquired stenosis may be the result of chronic gastritis, or develop independently, sometimes as part of a general proliferation of connective tissue.¹ If to the physical signs of tumor of the pylorus be added the signs and symptoms of dilatation, we have the clinical picture of hypertrophic stenosis of the pylorus. It is extremely rare to find complete obstruction.

Congenital hypertrophic stenosis, as Metzler and Caudley point out, has for its characteristic features: (1) vomiting, occurring without apparent cause and persisting in spite of treatment; (2) the absence of bile from the vomited matter; (3) obstinate constipation; (4) marasmus; (5) the presence of a tumor in the region of the pylorus; (6) the absence of abdominal distention except from dilatation of the stomach itself in some instances; and (7) the absence of signs or symptoms of gastritis and of the more common forms of intestinal obstruction. The diagnosis depends entirely on the characteristic symptoms arising during the first few weeks of life and on the presence of a tumor.

DISEASES OF THE STOMACH CHARACTERIZED BY PAIN AND VOMITING.

Cancer of the Stomach.

The clinical symptoms are varied. Gastric cancer may occur without any symptoms whatever, and be discovered after death from other causes. On the other hand, general marasmus and cachexia may be present without local symptoms. In some cases the gastric symptoms are slight and obscured by the symptoms of secondary growth in the liver or peritoneum.

Some cases of carcinoma may give a history pointing to prolonged chronic gastritis, but the most common and distinctive feature is a sudden onset of gastric symptoms without evident cause in a person beyond middle life. Loss of appetite is marked, and in spite of careful treatment there is loss of flesh and strength. The vomiting gradually becomes more frequent. The general appearance of the vomitus is at first like that of chronic gastritis. Soon it becomes streaked with blood, or a moderately large hemorrhage may take place. The vomited matter is often dark in color, like coffee-grounds in appearance. The relation of vomiting to the time of taking meals depends upon the seat of the disease. If it is at the cardiac end of the stomach, the vomiting may take place at once; if in the greater curvature, within twenty minutes or one hour and a half after taking food; if at the pyloric orifice, the

¹ See author's case, Phila. Path. Soc. Trans., 1881-83, vol. xi., p. 216.

vomiting is delayed several hours. As the disease advances and obstruction becomes more complete at the cardiac orifice, food is immediately regurgitated, unless secondary dilatation of the œsophagus takes place. When there is gastric dilatation, the vomiting may take place at longer intervals and be characteristic of the vomitus of dilatation. Constipation is the rule.

Tumor. After the symptoms of chronic gastritis have continued for some time without relief, a tumor may be detected if it is sufficiently large and is situated favorably for examination. (See Tumors of Abdomen.) If the growth is situated at the cardiac orifice of the stomach or on the lesser curvature, it is often impossible to detect it; if at the pyloric orifice, the tumor is found to the right of the median line above the umbilicus, but may be forced down by the weight of the stomach and felt at the umbilicus. (See Plate XL., Fig. 2.) When dilatation follows pyloric tumor, it may be still lower down, as in a case of the writer's, in which it was found two inches below and to the right of the umbilicus. In tumor of the greater curvature the mass is detected below the margin of the ribs on the left side, and may be as low down as the umbilicus. If the greater curvature is involved, the organ may contract, and hence the physical signs indicating the lower border of the stomach are found higher up than in health.

It is necessary to exclude tumors due to other causes. This is sometimes difficult—indeed, so far as the location and physical characters are concerned, often impossible. The most pronounced diagnostic feature of tumor of the pylorus is the occurrence of secondary dilatation of the stomach. For a differential diagnosis of tumors in this region, see Palpation of Abdomen.

Symptoms due to Metastasis. The *liver* is the most frequent seat of secondary growths. The organ enlarges, and its surface becomes covered with nodules. (See Plate XL., Fig. 2.) Jaundice occurs in rare instances. The enlarged liver may cover the stomach and hide the local mass. The *inguinal* glands may enlarge. At times there is enlargement of the *supraclavicular glands*, suggestive also of intra-abdominal carcinoma from other causes.

The general symptoms are those of *emaciation* and *cachexia*. The *emaciation* is extreme, and in some cases may be out of proportion to the local symptoms.

The symptoms of *cachexia* are those of emaciation and anæmia. The *anæmia* becomes profound. The pallor of the face is striking; often it is of a yellowish and straw-colored hue. It must not be confounded with jaundice—examination of the conjunctivæ is usually sufficient to distinguish the two. The skin is flabby, and the subcutaneous fat is entirely lost; the emaciation is not so marked as in cancer of the œsophagus, except when there is complete cardiac stricture. The nutrition of the skin suffers, boils are common, and ulcers may occur in the terminal stages. Subcutaneous hemorrhages are seen on the backs of the hands, and feet, and on the legs and arms. There is slight œdema over the ankles.

General atrophy of the internal organs takes place, so that the heart

becomes small; it loses its strength; the patient becomes weaker and weaker, the pulse rapid and feeble.

If *fever* occurs in the course of the disease, it is usually due to secondary accidents, as suppuration in a tumor, or perforation with septic peritonitis. The usual course of the temperature is normal until the latter stages, when it is subnormal.

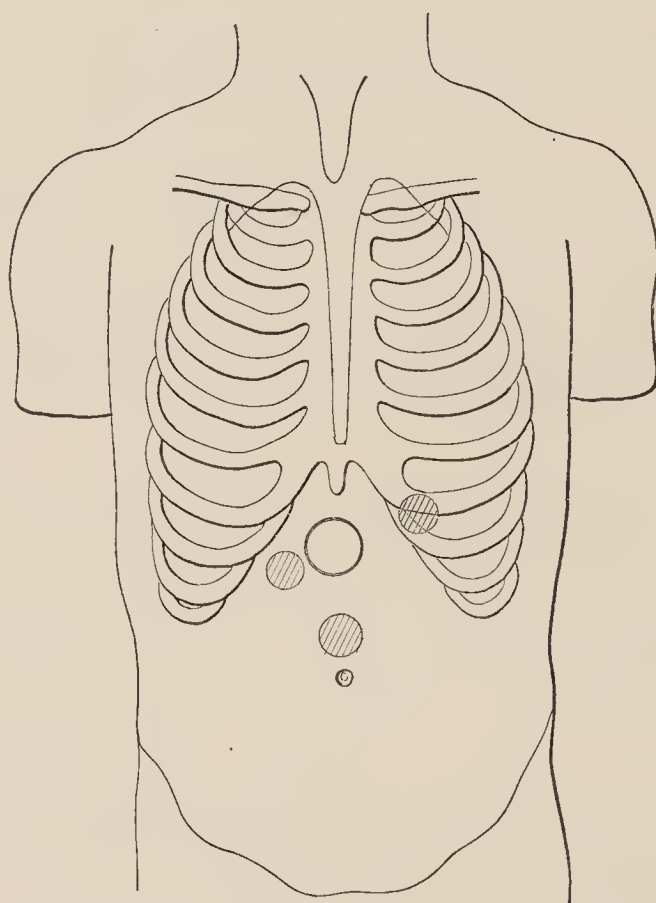
DIFFERENTIAL DIAGNOSIS OF GASTRIC CANCER, GASTRIC ULCER, AND CHRONIC GASTRITIS. (*Welch.*)

GASTRIC CANCER.	GASTRIC ULCER.	CHRONIC CATARRHAL GASTRITIS.
1. Tumor is present in three-fourths of the cases.	Tumor rare.	No tumor.
2. Rare under forty years of age.	May occur at any age after childhood. Over one-half of the cases under forty years of age.	May occur at any age.
3. Average duration about one year, rarely over two years.	Duration indefinite; may be for several years.	Duration indefinite.
4. Gastric hemorrhage frequent, but rarely profuse; most common in the cachectic stage.	Gastric hemorrhage less frequent than in cancer, but oftener profuse; not uncommon when the general health is but little impaired.	Gastric hemorrhage rare.
5. Vomiting often has the peculiarities of that of dilatation of the stomach.	Vomiting rarely referable to dilatation of the stomach, and then only in a late stage of the disease.	Vomiting may or may not be present.
6. Free hydrochloric acid usually absent from the gastric contents in cancerous dilatation of the stomach; lactic acid present.	Free hydrochloric acid usually present in excess in the gastric contents.	Free hydrochloric acid may be present or absent.
7. Cancerous fragments may be found in the washings from the stomach or in the vomit (rare).	Absent.	Absent.
8. Secondary cancers may be recognized in the liver, the peritoneum, the lymphatic glands, and, rarely, in other parts of the body.	Absent.	Absent.
9. Loss of flesh and strength and development of cachexia usually more marked and more rapid than in ulcer or in gastritis, and less explicable by the gastric symptoms.	Cachectic appearance usually less marked and of later occurrence than in cancer, and more manifestly dependent upon the gastric disorders.	When uncomplicated, usually no appearance of cachexia.
10. Epigastric pain is often more continuous, less dependent upon taking food, less relieved by vomiting, and less localized than in ulcer.	Pain is often paroxysmal, more influenced by taking food, oftener relieved by vomiting, and more sharply localized than in cancer.	The pain or distress induced by taking food is usually less severe than in cancer or ulcer. Fixed points of tenderness usually absent.
11. Causation not known.	Causation not known.	Often referable to some known cause, such as abuse of alcohol, gormandizing, and certain diseases, as phthisis, Bright's disease, cirrhosis of the liver, etc.
12. No improvement, or only temporary improvement, in the course of the disease.	Sometimes a history of one or more previous similar attacks. The course may be irregular and intermittent. Usually marked improvement by regulation of diet.	May be a history of previous similar attacks. More amenable to regulation of diet than is cancer.

Examination of the Stomach Contents. Hydrochloric acid is absent in most instances, its absence depending largely but apparently not entirely on an associated atrophy of the gastric tubules. Lactic acid, on the other hand, is commonly present even in the earliest stages, and when associated with absence of HCl is very diagnostic. For an accurate diagnosis repeated examinations must be made. (See page 595.) Other general and local conditions, as fevers on the one hand, or dilatation on the other, are attended by occasional absence of hydrochloric acid; in carcinoma it is the persistent absence of HCl and presence of lactic acid that are diagnostic. Other important signs are the presence of Oppler-Boas bacilli and, according to Strauss, of pus. Pepsin is usually diminished in this as well as in most other conditions almost in direct proportion to the diminution of HCl, while the milk-curdling ferment disappears last of all.

The Urine. Indican is increased in amount, acetone and diacetic acids may be present in the urine; otherwise there is no change.

FIG. 370.



Site of pain in gastric ulcer is marked at O.

Diagnosis. In the diagnosis of gastric cancer the following must be borne in mind: 1. The age of the patient. 2. The occurrence of causeless dyspepsia without relief. 3. Rapid loss of flesh and strength, with cachexia. 4. The occurrence of pain in the epigastrium, continuous, increased by food, but not relieved by vomiting as in ulcer, and not distinctly localized. 5. Tumor—hard, circumscribed, followed by the physical signs of dilatation, if at the pylorus. 6. Vomiting is necessarily associated with the taking of food, in which fragments of cancer may be found; blood-cells are common; they may be detected on microscopical examination, or by the test for hæmin. 7. Examination of stomach contents: (a) except in dilatation the fasting stomach is empty; (b) hydro-

chloric acid is often absent, whereas lactic acid is present ; (c) delayed absorption is present, indicated by motor tests. 8. Hemorrhage. In small amounts, usually of characteristic, coffee-grounds appearance. 9. Metastases—above the left clavicle, in the liver, in the inguinal glands, rarely in the lungs and peritoneum. 10. Eichhorst speaks of persistent itching of the skin and insomnia as characteristic symptoms. 11. Finally, the comparatively short duration of the case ; rarely does it extend over a period of more than two years.

Cases of cancer of the stomach may present only symptoms of anæmia. In this manner the disease has been confounded with *pernicious anæmia*. The most important distinction seems to be, as pointed out by Henry, that in cancer the red cells never fall below 1,500,000 per cubic millimetre, while in pernicious anæmia they practically always do at some stage of the disease. The diagnosis is, however, at times almost impossible.

Ulcer of the Stomach.

Simple round ulcer of the stomach may occur at any age, but is most common in young anæmic women. It may be the result of an erosion of hemorrhagic infarcts by the gastric juice.

Symptoms. The symptoms are variable. The cases have been divided by Welch into four classes : (1) those in which there are no symptoms whatever, the ulcer being found after death from other diseases ; (2) no symptoms until the sudden occurrence of hemorrhage or perforation ; (3) the symptoms of chronic gastritis or gastralgia only ; (4) typical cases, with the characteristic symptoms, *pain, hemorrhage, and vomiting*. The symptoms of gastric ulcer may develop suddenly.

Pain. The pain is localized ; it is usually confined to a small area in the epigastrium. It may be seated behind the cartilages of the sixth and seventh ribs, or may be complained of in the back, between the eighth and ninth dorsal, and extending as low down as the first and second lumbar vertebræ. It is of a burning or gnawing character, is increased by eating, and comes on in from two to ten minutes after the ingestion of food. It is relieved by vomiting, or after the act of digestion is completed ; but a persistent dull pain or a feeling of soreness remains. In addition to the ordinary pains, there may be attacks of gastralgia. The pain is increased by pressure. It may be modified by the position of the patient. It may be relieved by lying on the back when the ulcer is in the anterior wall ; or by lying on the abdomen when in the posterior wall. Boas has pointed out that there is in many cases, beside the extreme epigastric tenderness, a point of great tenderness in the back, on the left side near the spines of the tenth to the twelfth dorsal vertebræ.

Vomiting. Vomiting occurs shortly after the ingestion of food. It is not attended by retching. The vomited matter may contain blood.

The vomited matter and the contents of the stomach contain hydrochloric acid, which may be in excess. Eichhorst thinks it is always in excess.

Hemorrhage. Blood in the vomitus gives the latter a brown or reddish color. It may be detected by the usual methods. Hemorrhage may

occur, however, independently of the act of vomiting. It varies in amount from half a pint to a quart. It may be so severe as to cause collapse. Sometimes, instead of being discharged as a profuse hemorrhage, the blood may gradually ooze from the ulcer and collect in the stomach before being vomited. It is then altered by the acid gastric juice. Sometimes the blood is not vomited, but passed in the stool, which is then tarry. Tarry stools also follow the vomiting of blood. In the course of ulcer a hemorrhage may be so severe that death takes place before vomiting occurs. The stomach is then found to be filled with blood.

The stomach bougie should not be used after hemorrhage or when there is good reason to suspect the existence of an ulcer; the nature of the contents must be determined by an examination of the vomited matter.

It is always very important to determine that blood supposed to have been vomited has not come from the lungs. In the latter case the blood is usually bright red and frothy, the lungs contain moist râles, and usually show some of the physical signs of tuberculosis.

A blood-count in cases of ulcer often shows a chloro-anæmia, particularly after hemorrhage. This is not distinctive of ulcer, however, as it is seen always in chlorosis and in many cases of phthisis, secondary syphilis, and in other conditions.

General Symptoms. In a case of long standing, the face is anxious and the lines are sharpened. If there is much hemorrhage, anæmia ensues. There is not much wasting and no fever. Chronic dyspepsia and constipation may be present during the intervals in which the severe symptoms are in abeyance. The period of abeyance varies in duration, and the symptoms may come on without cause, as in gastric crises, during which time the vomiting may persist for two or three days. I saw a girl of twenty years with most severe gastric hemorrhage and classical symptoms of ulcer. With careful treatment she improved. After marriage she remained well until pregnancy. During the first periods of this condition vomiting was extreme; it then subsided, whereupon, without warning, a gastric crisis took place. The vomiting of blood continued for many days, and the symptoms of gastric ulcer remained for a month.

One of the characteristic features of the disease is the recurrence of symptoms after a long period of abeyance. A patient under my care during the last ten years has had three undoubted attacks. It is possible that during each period ulcers healed, to be followed after a time by the development of new ulcers.

Diagnosis. The diagnostic features are: 1. The age. 2. The long duration. 3. The occurrence of emaciation up to a certain point only; most of the patients are underweight and have a gaunt look, particularly males. 4. The characteristic pain. 5. The vomiting. 6. The hemorrhage. 7. The periods of relief from symptoms. 8. The absence of marked nervous symptoms which attend gastric neuroses. 9. The absence of dilatation of the stomach. 10. The hyperacidity of the gastric juice.

The Accidents of Ulcer of the Stomach. 1. The occurrence of perforation. Sudden severe pain, with collapse. The pain is usually in

the epigastrium, but may be in the back as high as the seventh or eighth dorsal vertebra.

2. Hemorrhage, which may cause death immediately, with either vomiting of blood or retention in the stomach.

3. With healing of the ulcer, stenosis at the pyloric orifice may take place, with subsequent dilatation of the stomach.

Syphilis of the Stomach.

Until recently syphilis of the stomach has been generally considered to be extremely rare, but records of its discovery post mortem by Flexner and others, and of well-authenticated clinical cases, chiefly reported by Dieulafoy, Einhorn, and Dalglish, have lately been so numerous as to make it evident that it must always be held in mind in cases of ulcer that are rebellious to treatment, or in the presence of a mass that does not show distinct characteristics of cancer. Syphilis, clinically, usually presents the signs of ulcer or of a mass which is likely to be mistaken for carcinoma. The diagnosis of syphilitic ulcer depends upon a history of syphilis, or the presence of other signs of the disease, the lack of success with the usual treatment and the rapid results with specific treatment.

The distinction between syphilis and cancer can be made only tentatively until the effect of treatment is seen. The main points are a history of syphilis, absence of a decided cancerous cachexia, and the effects of treatment.

Einhorn has described cases of syphilitic stenosis of the pylorus in which the diagnosis was made by the points mentioned.

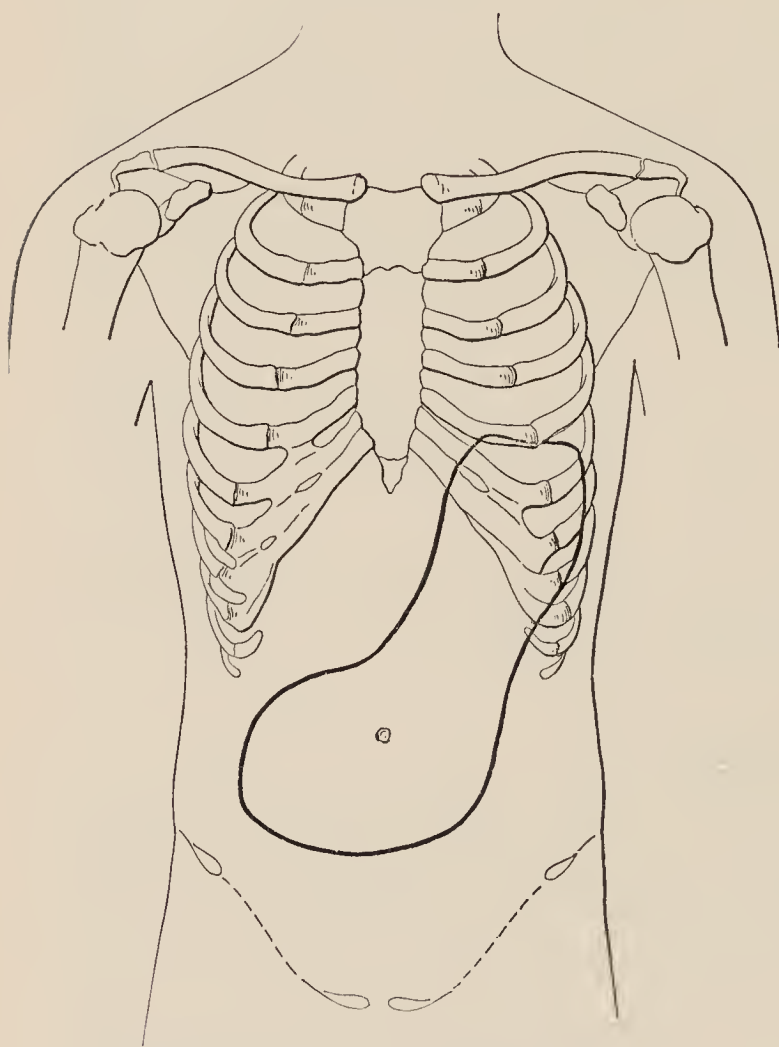
Gastroptosis.

Although downward displacement of the stomach from its normal position in the epigastrium forms part of the general symptom-complex known as enteroptosis (*q. v.*), Glénard's disease, or descent of all the abdominal viscera and one or both kidneys, competent authorities have recently cast a doubt on the propriety of assuming displacement of the colon in all cases in which the existence of gastroptosis has been demonstrated by inflation, unless the intestines themselves have been distended and thus shown to be involved in the general descent of the abdominal contents. A good deal of study has recently been bestowed on the subject, notably in this country, with the result of showing the great importance of recognizing the condition, and the possibilities of rational therapeutic measures for its relief.

Etiology. A number of theories have been advanced, some to be speedily discarded, to explain the causation of gastroptosis. Dismissing those that have been found untenable, such as the presence of adhesions between viscera, primary descent of the colon—coloptosis, congenital predisposition, which play a certain part but fail to explain more than a small proportion of the cases, it may be stated that the underlying causal condition is "anything that constricts the thorax and enlarges the abdominal cavity" (Steele and Francine), in other words, pressure on the

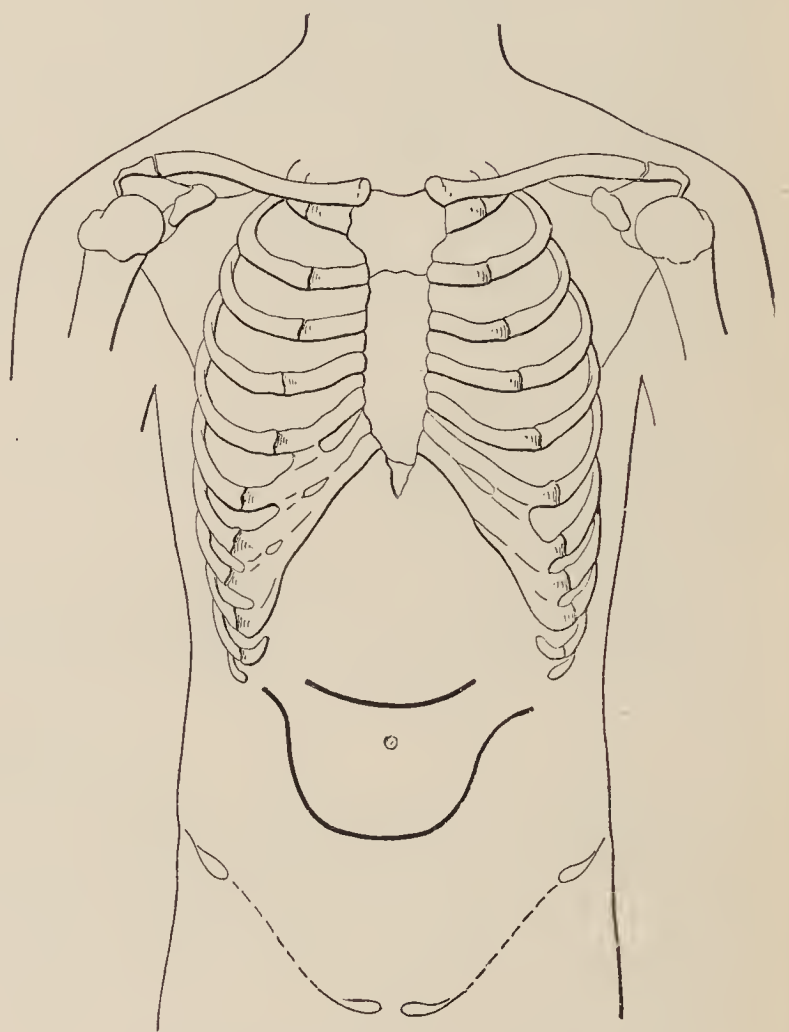
stomach from above, or removal of the support from below by the sinking down of the contents of the lower half of the abdomen. A variety of factors may bring about such a state of affairs: tight lacing and the pressure of the waist-band, pressure from a neoplasm of the liver, dilatation and pyloric obstruction, general muscular debility and emaciation after an acute systemic disease, relaxation of the abdominal walls and diastasis of the recti (Webster) after repeated pregnancies, and finally, traumatism and sudden muscular over-exertion. Steele divides the cases into primary gastropotosis with pyloric dilatation and secondary gastropotosis with general dilatation, the gastrectasis in the latter group being probably the basal condition.

FIG. 371.



Dilatation with pyloric obstruction.

FIG. 372.



Gastropotosis, with general dilatation.

It is evident from the causes enumerated that women are the chief sufferers from the disease; but as the available statistics are hardly numerous enough to furnish a trustworthy basis for calculation, and the condition may, in addition, exist without producing symptoms, the incidence is difficult to determine. The proportion as regards sex is probably about 7 to 1 in favor of women, except in the secondary and much rarer form, gastropotosis with general dilatation, in which the relation is reversed. The disease is most common in early adult and middle life; but the fact that a few cases in children have been reported by Hemmeter would seem to indicate that more careful physical examination might result in a considerable lowering of the age limit. Persons whose occupations require them to stand for a number of consecutive hours, as saleswomen, and motormen and conductors on trolley-

cars, the constant jarring of which is possibly a contributory factor, appear to possess a special tendency to the disorder.

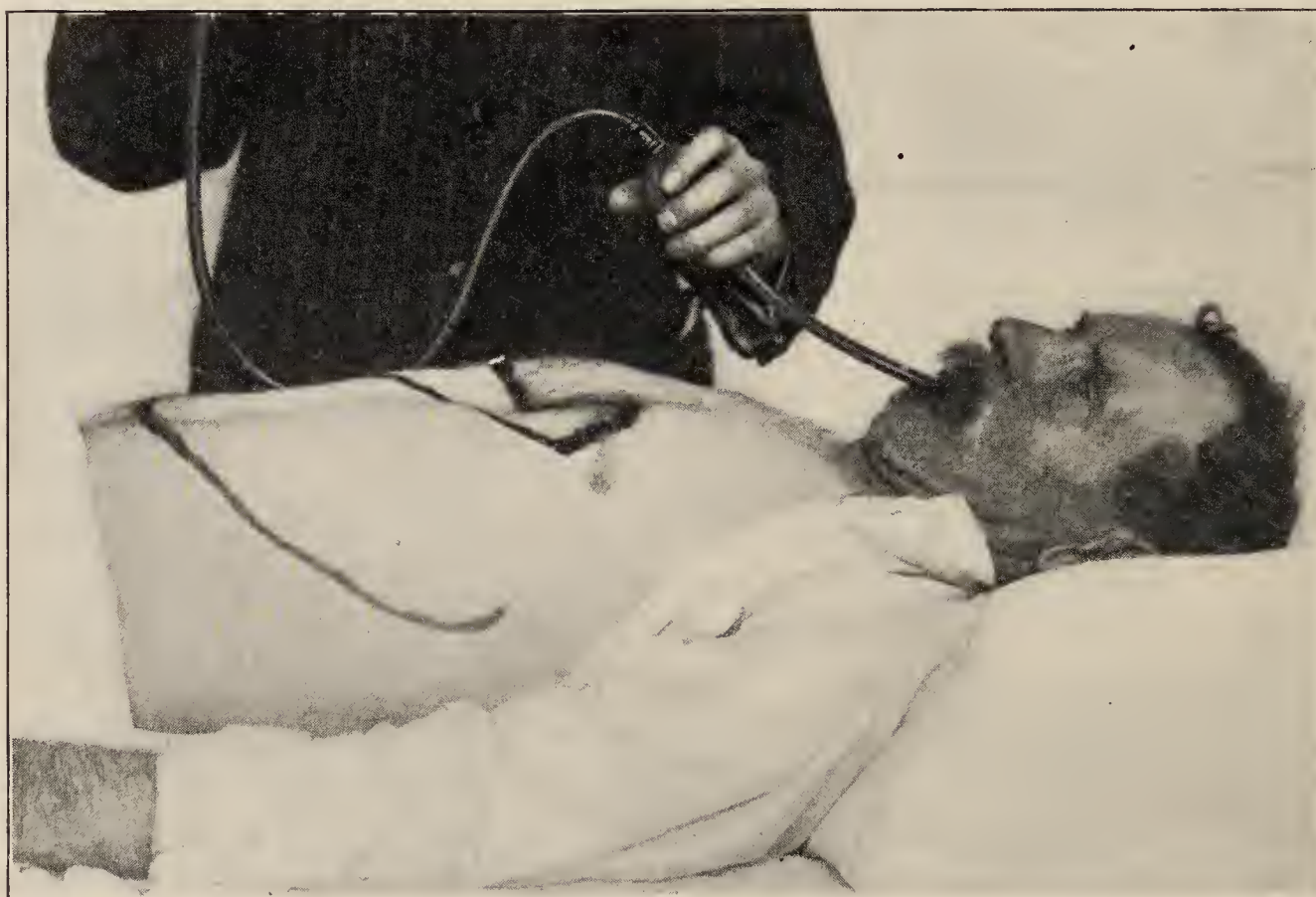
Symptoms. It is to be remembered that gastropotosis may exist without producing any subjective symptoms, its presence being discovered accidentally by a routine physical examination or at the autopsy. In many cases the dominant features are those of the accompanying neurasthenia, which should be interpreted as secondary to the anatomical disturbance, as undoubted cases of gastropotosis have been reported by competent observers, especially in men, in which there was no suspicion of neurasthenia. Sooner or later in the course of the disease the effects of dilatation and deficient motor power make themselves felt, and it is for the relief of symptoms pointing to these conditions that the patients usually present themselves. They complain of fulness in the epigastrium, distress and flatulence after meals; nausea, eructations of gas, and pyrosis, sometimes going on to palpitation of the heart and dyspnoea; headache, malaise, somnolence, inability to work and other symptoms of auto-intoxication. Pain is not a prominent or even a constant feature, and when present is probably due to some morbid process within the stomach; one theory that has been advanced is that it is the expression of an insult to the solar plexus. In cases marked by extreme dilatation with fermentation of the gastric contents, the overloaded viscus may seek relief in vomiting; otherwise, however, that symptom is not as a rule complained of. Finally the patients complain of a train of symptoms that strongly resemble the familiar features of uterine displacement, a sense of dragging and weight with sacral pain, which not infrequently are attributed to some gynecological disorder. According to Steele, chronic jaundice is an occasional accompaniment of gastropotosis and appears to be due to the presence of adhesions causing kinking and obstruction of the bile-ducts.

Physical Signs. The patients are usually emaciated and more or less anæmic, the chlorotic type predominating. The frequent association of chlorosis and gastropotosis led Meinert to the theory that there is a causal connection between the two conditions, and that all cases of chlorosis are primarily caused by descent of the stomach, but the theory is now regarded as untenable. The belly in most cases is flat; when the relaxation, which is quite characteristic, is extreme, the abdomen may be pendulous; in men, however, the tissues of the body may be quite firm. The epigastrium is flattened and there is a prominence somewhere below the umbilicus corresponding to the abnormal position of the stomach. The pulsations of the abdominal aorta can usually be felt with ease and may be perceptible to the patient. The transverse band that is felt running across the abdomen is now generally believed to correspond to the pancreas: indeed, its identity with that structure was recently demonstrated at an operation by W. F. Hamilton. Other signs of enteropotosis, Glénard's belt sign, diastasis of the recti muscles, and others that will be referred to in the proper place, should be looked for.

Inflation. For a positive diagnosis of gastropotosis the physical demonstration of the gastric area in an abnormal position, by actual distention of the viscus with a gaseous medium supplemented by percussion and

auscultatory percussion, is absolutely indispensable, and the most practicable as well as the most effective means of accomplishing this object is by simple inflation through a stomach-tube with an ordinary atomizer bulb. The carbon dioxide method has the sole advantage of enabling one to dispense with the introduction of a stomach-tube, and the distinct disadvantage that the gas rapidly escapes through the œsophagus and into the small intestine, so that the distention does not last long enough for a thorough examination to be made. With ordinary inflation, if the air is pumped in rapidly at first, spasm is induced at the pylorus and prevents the premature escape of the distending medium into the small intestine. (Pepper and Stengel.) The stomach should first be emptied in the usual manner, after which an atomizer bulb is fitted to the stomach-tube, without removing the latter, and the distention carried to a point just short

FIG. 373.



Inflation of stomach.

of causing discomfort. Owing to the lax condition of the abdominal walls, the outline of the stomach is usually at once visible, or it can be determined by palpation, or, as some authorities advise, by auscultatory percussion. The percussion-note over the distended viscus is of course tympanitic. The following sign may be sufficient: "when the bulb of the syringe is compressed, forcing the air through the tube, a peculiar metallic ring can be heard with the stethoscope over the stomach. When the bell of the instrument is moved along beyond the stomach limits, the sound instantly loses its metallic character. The same authority advises that the air be allowed to escape before the tube is withdrawn, in order to prevent the distress which often accompanies the escape of the air from the stomach, probably on account of a spasm at the cardiac orifice. When gastroptosis is present, the greater curvature is found below the level of the umbilicus, and in extreme cases may

extend almost to the pubes. In total descent of the stomach the entire outline occupies a position below the median transverse line of the body.

Blood. Except for the frequent presence of chlorosis already referred to, or a mild simple anæmia, there are no changes in the blood-count.

Urine. Examination of the urine may reveal the presence of constituents pointing to intestinal fermentation, otherwise the secretion is normal.

Gastric Contents. The findings are not distinctive and probably have no connection with the gastropptosis; the chief value of an analysis is derived from the therapeutic indications which it affords.

Diagnosis. In the light of recent researches gastropptosis must be regarded as a much more common condition than was formerly supposed, and it is therefore important to make a thorough physieal examination of all cases presenting the symptoms of dilatation and deficiency of motor power, which include most cases of so-called gastric neurasthenia. If inflation is resorted to, a mistake in diagnosis is hardly conceivable. The condition for which gastropptosis is most likely to be mistaken, and with which indeed it is frequently associated, is displacement of the womb; in many supposed gynecological cases a complete cessation of the reflex nervous symptoms might be brought about by rational treatment directed to the correction of a displacement of the abdominal rather than of the pelvic viscera.

DISEASES OF THE INTESTINES.

The intestine is a canal of varying dimensions, the physiological office of which is to propel material received from the stomach and to permit of the digestion and absorption of that which is to serve for the nutrition of the body. The canal is richly supplied with bloodvessels and lymphatics. It is made up of mucous membrane, muscle, and peritoneum. For the purpose of digestion fluids are secreted either from the intestinal glands or from the large neighboring glands which discharge into the canal. Diseases that affect the canal impair or cause an abeyance of the physiological offices. As these offices—absorption and digestion—are essential to nutrition, it is not surprising that the body-weight and strength are impaired. We know too little about the function of digestion to utilize such knowledge in diagnosis. Intestinal digestion is also dependent upon the healthy performance of the functions of the liver and pancreas. It is difficult to draw fine lines of distinction even in health, and intestinal pathology is closely interwoven with hepatic and pancreatic pathology.

Alterations of the function of the intestine as a canal give rise to distinctive symptoms. Either its movements are too frequent and rapid, causing *diarrhœa*, or too sluggish, causing *constipation*. Obstruction of the canal leads to symptoms common to such a condition (see Morbid Proceess), modified by the physiological duties and the anatomical structure of the canal.

The morbid processes are hyperæmia, inflammation, the degenerations, and new growths. The symptoms that attend these processes are not

different from the symptoms that attend such processes in similar structures elsewhere. It must not be forgotten that the function of the canal is influenced by each process. On account of the process we may have *pain* and *fever*; on account of the impaired function, *pain*, *flatulence*, *diarrhœa* or *constipation*, *change in the character of the stools*, and *impaired nutrition*. Some of the above morbid processes may lead to the mechanical condition, *obstruction*.

The morbid alterations of the intestinal tract are ascertained from data obtained by *inquiry* and by *observation*. The data obtained by inquiry include the subjective symptoms—*pain*, and discomfort from flatulence. By observation the general condition of the patient, the presence of tenderness, alterations in the size and shape of the abdomen, and other physical phenomena are observed. The feces are carefully studied for the purpose of determining functional disturbances in the bowel, the presence of ingredients due to some morbid process, as serum, blood, pus, or mucus, or of extraneous matter, as worms or foreign substances. The feces are studied with the naked eye, with the microscope, and by bacteriological methods.

One symptom may be the chief manifestation of a disease, as *pain* in lead colic, *diarrhœa* in several morbid disorders, *constipation* in others. In the discussion of the special symptoms a consideration of the diseases of which the symptom is the main expression will be taken up.

Parasites. The intestine is the recipient of material for nutrition. Parasitic forms of animal life, or their ova or spores, may enter the intestine with the food. They either remain in the intestinal tract or wander into other structures. They include animal and vegetable organisms, such as forms of protozoa, vermes, and fungi. While the canal is open to infection by various micro-organisms, it is the natural habitat of others, which may become deleterious agencies when the conditions of their environment are changed. Thus *Bacillus coli communis* is, in man with normal epithelial structure and normal secretions, an innocuous parasite which, when inflammation sets in, may become virulent.

The symptoms produced by the *protozoa* and *fungi*, or by their products, the ptomaines, are of an infectious or toxic nature. Inflammation is produced locally.

The symptoms produced by *worms* in the intestinal canal, are: (1) reflex; (2) symptoms due to catarrhal inflammation; (3) symptoms due to the action of the parasite on the blood—anæmia; (4) symptoms due to wandering of the parasite, as in trichinosis. (See Feces.)

SYMPTOMS OF THE TÆNIÆ AND BOTHRIOCEPHALI. There may be no symptoms save discharge of the parasite or portions of it by the rectum. In other cases the symptoms of intestinal dyspepsia or intestinal catarrh are observed. Headache, giddiness, lassitude, and itching at the nose and at the anus are said to be present. The patient becomes hypochondriacal. Convulsive disorders occur. Hysteria, forms of epilepsy, grinding of the teeth at night, and restlessness attend the presence of the parasite in the intestine. In all convulsive disorders the possibility of worms as a cause must be remembered.

SYMPTOMS OF ASCARIDES. (1) Gastro-intestinal catarrh; (2) symp-

toms of obstruction (rare); (3) symptoms due to wandering—as to the hepatic duct, to the stomach, or to the vagina; (4) nervous symptoms of reflex origin; (5) the worm or its ova in the feces.

SYMPTOMS OF OXYURIS VERMICULARIS. (1) Gastro-intestinal dyspepsia or catarrh; (2) itching or heat at the anus, worse in bed; (3) vesical and rectal tenesmus; (4) erythema about the anus; (5) priapism; (6) vulvitis and vaginitis; (7) the worms in the feces.

THE STRONGYLUS. The symptoms are local, with the symptoms of profound anæmia. The discovery of the ova in the feces distinguishes this form of anæmia from other varieties.

UNCINARIA DUODENALE. The symptoms are those of grave secondary anæmia and of chronic gastritis.

The symptoms due to the presence of *Trichina spiralis* and filaria are discussed in their appropriate sections. (See Blood and Infectious Diseases.)

The Intestines in Other Diseases. The relationship of intestinal disorders to affections of other viscera will be discussed with each symptom. It must not be forgotten that derangement of this tract may have its origin in local causes or in causes remote from the intestinal tract, or in some general condition of the individual. Thus diarrhœa may be due to inflammation which is primarily local, or which may be secondary to infection. Nothing is more common than to see diarrhœa in a general infection such as septicæmia. In exophthalmic goitre the diarrhœa is not due to a local cause, but to some as yet unknown nerve disorder. Constipation may be due to central brain disease, to a general condition like diabetes, or it may have a local origin.

It must be remembered that the diagnosis of an intestinal lesion is never complete without determining its causes. Thus enteritis and ulceration occur in typhoid fever, in cholera, and in other infectious disorders, all of which are to be passed in review in making up a diagnosis. Diarrhœa is a symptom in Bright's disease, and the causal relationship must always be borne in mind.

Differential Diagnosis. Intestinal disease or disorders are not usually confounded with disease of other structures. It is worthy of remark, as a fact that is sometimes overlooked, that symptoms of intestinal obstruction are frequently due to peritonitis. Tumors of the intestine must be distinguished from tumors of the peritonæum, the stomach, pancreas, and liver, and the uterus and ovaries. The history, the seat and physical character of the tumor, and the associate symptoms point to the true condition.

Embolism. The intestines are supplied by the mesenteric arteries. Their branches may become the seat of emboli. The symptoms are sudden pain, intestinal hemorrhage, and discharge of a portion of intestine. The patients are the subjects of atheroma or heart disease.

Intestinal Indigestion.

Intestinal indigestion is said to be due to alterations in or diminution of the bile, the pancreatic, or the intestinal secretion. It is almost always attended by gastric indigestion, and may not be readily distinguishable from it.

Acute Intestinal Indigestion. Acute intestinal indigestion is due to the irritation of food not properly digested in the stomach. It is attended with colic, flatulence, and borborygmi. Some fever may develop, and diarrhoea may ensue. In the mild forms the tongue is coated, there are loss of appetite and some general pains. There is epigastric distress or pain in the right upper quadrant. Flatulence and constipation occur. The stools are often clay-colored, or may not be changed. Slight jaundice occurs, and there is an abundance of lithates in the urine. Accompanying gastric indigestion modifies the symptoms slightly.

Chronic Intestinal Indigestion. The symptoms are more marked and pronounced in chronic intestinal indigestion. The *local symptoms* are as follows: Pain beginning from two to six hours after eating, and complained of in the region of the liver, below the sternum, or in the umbilical region. It is dull and continues two or three hours, or until the next meal is taken. There is some tenderness. With the pain there are tympanites, borborygmi, and a sense of fulness in the abdomen; the bowels are constipated, and the stools are hard and dry. The constipation alternates with diarrhoea, and undigested particles of food are passed. The appetite is not lost, but is variable. Hemorrhoids are often present.

The *general symptoms* are marked, and are referred to the nervous system and the condition of the blood. There are great depression and hypochondriasis. The patients sleep badly, suffer from unpleasant dreams and tinnitus aurium; there are spots before the eyes and more or less constant headache. They complain of pain in the back and limbs, and hyperæsthesia and anæsthesia are present. There is inaptitude for mental exertion. Frequently the patient has sudden attacks, apparently due to the action of toxins, as sudden fainting followed by collapse, or vertigo. During these attacks there are great palpitation and tachycardia. The extremities are cold, and there are cold sweats over the body. Independently of the attacks, the patient is subject to palpitation and some dyspnoea. The urine is always high-colored, acid in reaction, and full of urates and uric acid; oxalate of lime may be present, and there is usually albuminuria from the irritation of uric acid. The patient early becomes anæmic, because of the auto-intoxication and defective assimilation. There is some emaciation; in some cases the emaciation is rapid. The complexion is sallow. If there is an abundance of oxalates, the patient complains of weight and heaviness about the loins. It is always important in cases of chronic diarrhoea to look for achylia gastrica, particularly if the diarrhoeal passages tend to occur soon after taking food. The stools may in other cases contain much undigested fat, indicating probable pancreatic disease. On the other hand, with loss of appetite, furred tongue, frontal headache, and drowsiness, the stools may be clay-colored and the bowels costive; this indicates obstruction to the outflow of bile.

Appendicitis.

This is by far the most important affection of the intestinal tract. It is of frequent occurrence compared with intestinal obstruction, and if recognized in time, is amenable to relief in a very large percentage of the

cases ; whereas intestinal obstruction is more frequently fatal. We see 25 cases, at least, of appendicitis in all its forms to one case of any form of obstruction. Its importance therefore is readily recognized. Appendicitis occurs most frequently in the young—in the large proportion of cases in persons under thirty. I have seen it as early as two years of age, although from the fifteenth to the thirtieth year it is more frequent than at any other period. The symptoms vary, but clinically may be divided into those of appendicitis without perforation and appendicitis with perforation. Appendicitis without perforation is characterized by relapses, and is known also as *recurring appendicitis*.

Appendicitis without Perforation. Cases of catarrhal appendicitis probably occur, although I am not prepared to say that catarrhal inflammation of the appendix gives rise to marked local symptoms ; for in cases on the post-mortem table in which the lesions of catarrh were found, there had not been any symptoms during life, either due to intestinal catarrh or pointing to appendicitis in any form. Moreover, many cases in which the attacks of appendicitis had at first been slight, finally developed into appendicitis with perforation. In the milder cases, if operative measures are resorted to during the intervals between the attacks, the appendix is always found to contain a fluid loaded with micro-organisms capable of causing purulent inflammation, as the staphylococcus or streptococcus. Clinically, therefore, all forms of appendicitis should be considered infectious, with, on the one hand, escape of the contents into the bowel, and natural relief of the symptoms ; or, on the other, complete obstruction with perforation. After removal of the appendix in cases of recurring appendicitis, I have always found pus or a mucopurulent material which was charged with streptococci or staphylococci, as well as *Bacillus coli communis*, natural to the intestinal canal in this region.

Symptoms of the Attack. After exposure to cold rarely, frequently after an indiscretion in diet, the patient is seized with pain referred to the right lower quadrant of the abdomen. It is paroxysmal in character, increasing in intensity, and may be described by the patient as colicky. The pain is usually such as to require the patient to take to bed and attempt to obtain relief by local applications ; but it may be so slight that the patient pays but little attention to it. He may even go about his business during the attack and seek professional advice at the office of a physician. Such cases as these are usually diagnosticated as ordinary cholera morbus or intestinal indigestion. The attack may be only moderately severe, particularly if there is diarrhœa. With the onset of the pain vomiting usually occurs. The bowels may be open or they may be confined. Vomiting may not occur if there is diarrhœa, but when present, is usually attended by some nausea, although this is not marked ; the vomiting is complete, there is no retching. It occurs at intervals, between which there is comparative comfort ; first, the contents of the stomach, and then mucus are ejected. If the patients are to get well, vomiting does not return unless excited by food. If peritonitis supervenes, vomiting returns in the course of two or three days. In bed the patient lies on his back with his right leg flexed.

Even with a mild degree of pain the skin is hot and the temperature slightly raised. In the cases in which the pain is more severe the general reaction is greater, the temperature rises rapidly to 102° or 103° F. The skin is hot and dry, the face flushed. The pulse is full and strong and in a young adult rises to 90 and 95. In some cases the patient complains more of the fever than of the pain after the first severity of the attack has subsided. The tongue is coated and the appetite is lost.

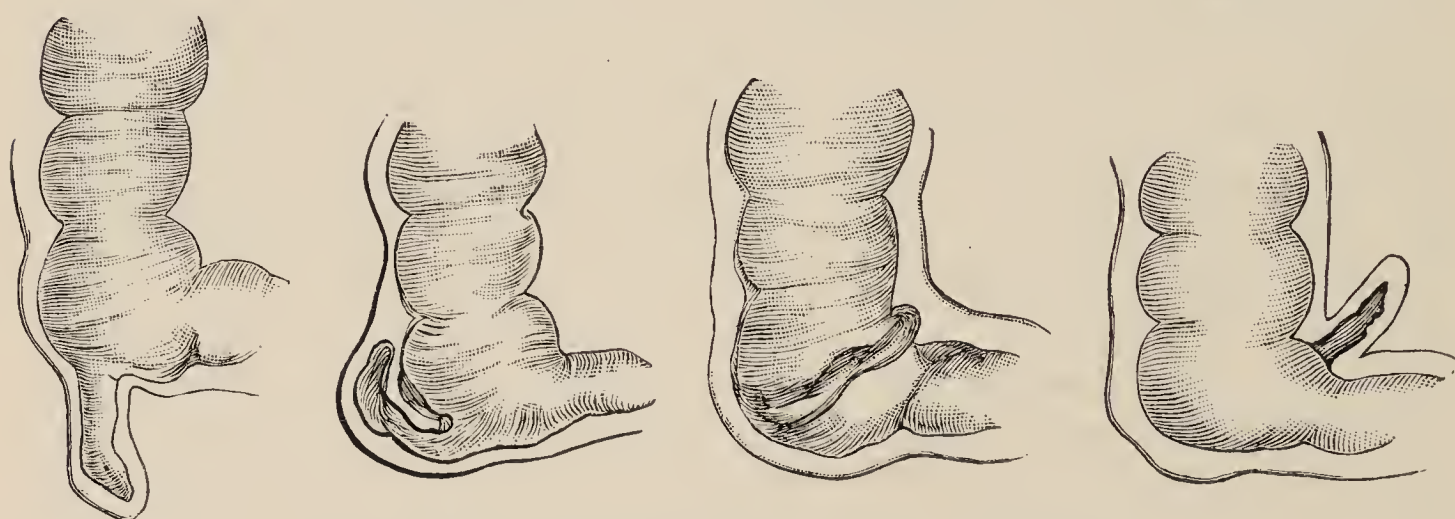
On *physical examination* the area which was the seat of pain is found to be tender. When examined with the tip of the finger pressing firmly, more marked tenderness can usually be found at a point midway between the anterior superior spine of the ilium and the umbilicus, known as McBurney's point, which corresponds to the site of the diseased appendix. The swollen tender appendix may occasionally be palpable. On *inspection* the affected area is slightly or may be considerably enlarged.

FIG. 374.

FIG. 375.

FIG. 376.

FIG. 377.



Some positions of appendix and relation to peritoneum.

Comparison must be made with the opposite side. It will be seen that the usual depression in front of the anterior spine, or the cavity toward the loin, is not so deep as on the opposite side. In front the surface may be even with the plane of the ilium. On *palpation*, in addition to tenderness and pain at the point previously indicated, fulness and enlargement can be distinguished. There are resistance to pressure and more or less rigidity of the abdominal muscles. On careful *measurement* the semi-circumference will be found in most instances to be larger than the semi-circumference of the opposite side. When bimanual palpation is performed, the left hand being placed in the loin behind and the right over the abdominal surface, resistance, induration, and rigidity can more easily be detected. On *percussion* there is change in the note compared with that of the opposite side, and change in the percussion-note during the course of the disease. This is particularly the case if the symptoms go on to perforation. On careful deep percussion a dull tympanitic tone is elicited, or a distinct area of dulness can be mapped out, but in some instances the distended cæcum yields tympany, which is greater than on the opposite side.

The *pain* is usually referred to the region above mentioned. The pain may be in the lower quadrant on the *left* side instead of the right, in those cases in which the appendix dips into the pelvis. It may also be

referred to the bladder or genitals, and be attended with vesical tenesmus and frequent micturition. The character of the pain and the bladder symptoms are such as to simulate an attack of renal colic, with the passage of sand. On account of the location the pain may be attributed to the Fallopian tube or ovary, and thought to be due either to disease of those organs or to dysmenorrhœa. It is not likely to be mistaken for the pain of dysmenorrhœa if the patient is subject to pain at the usual monthly period. If, however, the physiological and the pathological congestion should take place at the same time, a mistake in diagnosis might occur, particularly as increased abdominal pain may cause a uterine discharge. The occurrence of fever would exclude dysmenorrhœa in cases in which this symptom was present. The pain and leg-flexion simulate hip-joint disease.

After the first twenty-four hours, during which the above-described symptoms take place, the fever continues. There is anorexia, but vomiting occurs only at long intervals if at all. The local symptoms continue, although usually modified by the treatment. Both general and local symptoms frequently subside after a free movement of the bowels, which occasionally takes place spontaneously. In other cases constipation continues a week or ten days, and even a longer period.

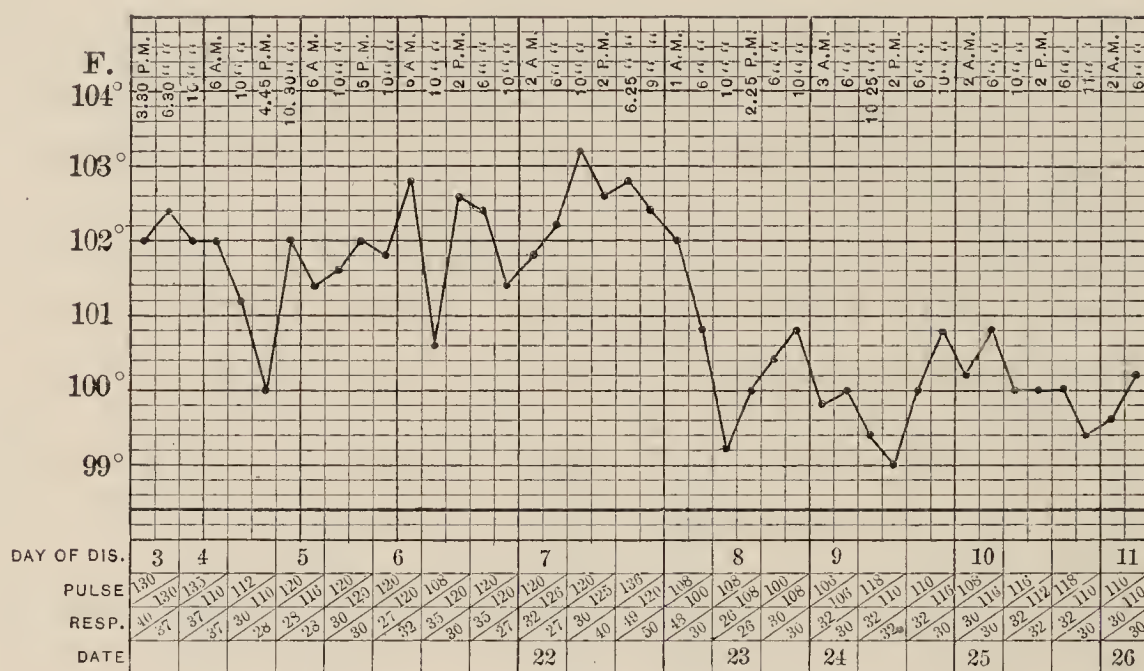
After five or six days at the furthest the fever subsides, the local distention lessens, the paroxysms of pain disappear, and convalescence ensues. Localized tenderness may, however, persist for a considerable period of time, and diarrhœa, or at least two or three evacuations each day, for a week or more. In rare instances peritonitis supervenes without the occurrence of perforation. Its onset under these circumstances is gradual, but the symptoms are like those of peritonitis under other circumstances, infection having taken place directly through the appendix.

When the fever continues, with mild diarrhœa, intestinal pain, and flatulence, the case may be mistaken for *typhoid fever*. The temperature is, however, more remittent in character in appendicitis, and the diarrhœa is not characteristic of typhoid fever. The characteristic eruption fever does not appear, the spleen is not enlarged, and the symptoms of the typhoid state do not ensue. The diazo reaction, the bacteriological examination of the stools, and the serum test may aid in forming a conclusion.

Recurrent Appendicitis. Frequent attacks of mild appendicitis occur; sometimes so frequently as every three months, or at intervals of a year. The attacks are similar to the attacks just described, although the duration is shorter. The local symptoms in some instances are more marked, because there has been a localized peritonitis previously. The induration is greater, and dulness more marked. In some instances the attacks are comparatively mild, continuing but twenty-four hours, and are described as attacks of colic. Often they are treated by the patient himself by household remedies alone. The patient spends a night in agony, with cramps, but the next day follows his usual habits. It is possible that there has been no fever with the attacks; but in all cases of recurrent appendicitis which I have seen, fever, although often slight, has been a constant accompaniment.

Appendicitis with Perforation. Before perforation takes place, the patient may have had symptoms of the mildest form of appendicitis for two or three days; or the attack may have extended over a long period of time, without any symptoms except colicky pain. As observations are not made, the presence of fever can not in such a case be utilized as a diagnostic feature. The perforation may take place early in the course

FIG. 378.



Acute appendicitis, with perforation and abscess. Female, aged eight years. Operation on seventh day.

of an acute attack, and result in *localized peritonitis* and *abscess*, or in *diffuse peritonitis*. If the latter, after the characteristic symptoms of appendicitis the symptoms of intense peritonitis set in. The abdomen rapidly becomes distended, the characteristic vomiting ensues, and collapse develops. Perforation under these circumstances has occurred within the first twenty-four or at most within forty-eight hours. Local inflammation about the appendix does not take place, and the local signs of an inflammatory tumor are not present, although tenderness at the special point can be elicited.

Abscess. If the perforation is more gradual, and there has been time for the occurrence of local inflammation about the appendix, by which pus is prevented from infecting the general peritoneum; or if perforation takes place behind, in the connective tissue which surrounds the mass, in which situation there is always inflammation, the local signs of abscess or of inflammatory tumor occur. There is swelling of the affected side; the normal outline is effaced. The area is indurated, and the early pronounced rigidity gradually gives way to a boggy sensation, with the appearance of œdema of the skin. This can be elicited by pressure over parts that are hard and resisting, as the spine of the ilium. Fluctuation can often be detected by bimanual palpation. Dulness is present, although in some instances it may be very slight, there being scarcely an appreciable change in pitch. Both light and deep percussion must be performed, and the note compared with the results of percussion over the opposite region. Palpatory percussion may alone indicate the departure

from normal. Examination per rectum may yield much information. An induration may be felt about the brim of the pelvis or in the rectal fossa, which fluctuates and may eventually soften. More accurate results may be obtained by bimanual palpation, with the index finger of one hand in the rectum and the other hand applied to the abdomen. Unless symptoms of general peritonitis with rapid infection of the system develop, the signs of abscess become more and more marked. The surface becomes reddened, and pointing may take place toward the groin or opposite the spine of the ilium. Sometimes the swelling increases in the direction of the loin, and the abscess may point in that situation.

As the abscess develops the general symptoms change. They now become the symptoms of *suppuration*. The fever is remitting or intermitting. There may be chills. Sweats are common, and there is loss of appetite and diarrhoea. Until recently it was customary, on account of the prevalent tardiness in operating, to see abscess develop in some other situation, or symptoms occur from burrowing of the pus in various directions. It may extend upward along the back of the colon, underneath the diaphragm, and thence to the pleura and lung, and be expectorated. The abscess may open into the rectum or into the bladder. If the local inflammation is virulent, even if general peritonitis has not taken place, the symptoms of *septicæmia* may rapidly ensue. This sometimes occurs quite early in the disease. There may be vomiting and septic diarrhoea, with slight delirium at night. An excessively rapid and feeble pulse is seen; in one instance it was irregular. Extreme prostration ensues, followed by symptoms of the typhoid state.

Gangrenous appendicitis is most treacherous. The early symptoms are like those of an acute attack; later all symptoms subside. Unless the temperature is taken or the physical examination is very painstaking, the patient is allowed to get up. The course may be afebrile. In a few days or a week an abscess forms about the slough, and then the usual phenomena of suppuration set in, or perforation may occur.

Diagnosis. It is clear that in cases of appendicitis we must attempt to recognize: (1) the inflammation before perforation has taken place; (2) the occurrence of perforation; (3) the occurrence of peritonitis due to either of the two conditions; (4) the occurrence of abscess (*paratyphlitis* and *perityphlitis*); and (5) the occurrence of *septicæmia*.

Appendicitis must be distinguished from *perinephric abscess* and the abscess which follows perforation of the intestine or cæcum at this point. *Perinephritis* can scarcely be distinguished from appendicitis unless there have been a previous history of renal calculus and pronounced evidence of disease of that organ preceding the formation of the abscess.

Hip-joint disease must be distinguished from appendicitis. The leg is flexed, the patient complains of pain about the region of the hip; unless careful observation has been made in the beginning of the attack, the early march of appendicitis may not be recognized. The two are confounded after abscess formation. The flexed leg of appendicitis can be extended under ether, and examination then shows the joint to be free from disease.

Fenwick says that *acute tuberculous peritonitis* may be confounded with perforation of the appendix. In both there may be pain and tenderness in the hypogastrium, dulness on percussion, and fever. In tuberculous peritonitis the onset is more gradual, the pain and tenderness more general, and there is no distinct tumor or increased tension in the hypogastrium. If there is dulness on percussion, the line generally varies with the position of the patient. Diarrhœa is urgent, and there are, in most cases, some signs of consolidation of the lungs. The absence of tumor in the right iliac region and in front of the rectum is the chief point of distinction; for when perforation occurs in phthisical subjects there is generally very slight pain, and severe diarrhœa is often the only prominent symptom. The appendicitis itself may be of *tuberculous origin*, as in several cases reported by the writer.

Inflammations of the Cæcum and Colon.

Typhlitis is an inflammation of the cæcum due to accumulation of fecal or foreign substances. The inflammation may remain as a localized enteritis, or may be followed by ulceration. In the majority of cases the ulceration is due to pressure by the contained foreign material or feces. The inflammation occurs in early life usually. The patients have been subject to constipation. The attack may follow some error in diet. There are pain in the right iliac fossa, constipation, and nausea. Moderate fever develops. On examination a fulness is discovered in the right iliac region, and the right thigh may be flexed; the cæcal region is tender to pressure, and a doughy sausage-shaped tumor may be outlined. The more severe symptoms last two or three days, but the local tenderness may continue a week or even longer. The tumor gradually disappears. If ulceration takes place, inflammation about the cæcum ensues. An abscess forms gradually in the flank. Perityphlitis is the term applied to this secondary abscess, although, as the term has been confused with paratyphlitis, it is better not to use it in this connection.

Pericæcal Abscess. Abscess about the head of the cæcum is due (1) to appendicitis, of which sufficient mention has been made; (2) to perforation of the cæcum, from typhlitis; (3) to perforation, on account of cancer of the intestine; (4) abscess secondary to kidney disease, perinephric abscess; (5) to abscess secondary to disease of the vertebræ. The physical signs are those of abscess due to perforation of the appendix. Abscess may occur behind the cæcum in cases of caries of the vertebræ and in some rare instances of empyema in which the pus has dissected downward. The symptoms are the local symptoms of abscess and the general symptoms of suppuration.

Fecal abscess, arising from ulceration of the colon, may be suspected, according to Fenwick, where there is a localized abdominal swelling, immovable in respiration or by a moderate amount of pressure with the fingers, the size and shape being altered when diarrhœa occurs, and when percussion over the tumor gives a tympanitic, or a more forcible stroke a dull sound, or when an emphysematous sensation is communicated to the fingers.

DISEASES CHARACTERIZED BY PAIN AND DIARRHŒA.

Acute Intestinal Catarrh.

Cause. Exposure to cold or the direct irritation of mechanical or chemical substances within the intestine. Irritating food that is not digested, or that can not be digested because of the quantity, tainted meats and unripe fruit usually excite an attack. Water saturated with impurities, or such as the individual is not accustomed to, may excite an attack. Strangers in a new locality are frequently subject to a diarrhœa until accustomed to the drinking-water, which in the native does not excite catarrh. Toxic substances, as poisons or drugs, or toxic substances the result of putrefaction, as ptomaines, are frequent exciting causes. Extension of inflammation from neighboring structures by infection, as in peritonitis, sets up a catarrh. Local diseases of the intestine, as ileus, intussusception, hernia, and ulcers of all forms are attended by catarrh of the intestine. It also occurs in cachectic states of the system, as cancer, anæmia, and Bright's disease. In disease of the heart and blood-vessels, or of the liver and spleen, where the disturbance of the circulation causes a congestion, catarrhal inflammation occurs. It is of common occurrence in the infectious diseases, and particularly in septicæmia and pyæmia.

Symptoms. *Diarrhœa* is the chief symptom, varying with the cause and the extent of the catarrhal inflammation. As has been previously indicated, the stools differ in frequency and color in the various types. They contain undigested matter; sometimes worms. Colicky pains about the umbilicus, with borborygmi and frequent desire to go to stool, attend each evacuation. The fever is of the remittent type, and is attended with some prostration. The urine is scanty and high colored. The symptoms vary somewhat with the location of the inflammation, although the exact locality can not be distinctly defined. Symptoms of proctitis, pain with tormina and tenesmus, however, enable the localization to be made to that portion of the bowel. These symptoms are more common than in inflammation apparently limited to the small intestine, while in colitis the violence of the rectal symptoms stands between that of enteritis and that of proctitis.

Diagnosis. The diagnosis of acute intestinal catarrh is not difficult. It is more difficult to determine the actual cause. If the attack occurs suddenly after the eating of improper food or the drinking of impure water, the irritation is probably due to that cause, and may be determined by the character of the feces. If they contain undigested food, the diarrhœa is probably due to indigestion. Catarrh from cold usually follows exposure, and is generally not very severe. To estimate the cause from poison or drugs, the condition of the rest of the intestinal tract must be investigated and other symptoms of poisoning must be inquired for. In arsenical poisoning there is always vomiting, and the discharges are of a choleraic nature. Collapse rapidly ensues. The other symptoms of arsenical poisoning must be inquired for and the history of exposure ascertained if possible. Intestinal catarrh due to infectious diseases is attended by the

symptoms of the respective affections, each of which is usually readily recognized. The intestinal catarrh which occurs on account of local disease of the bowel, as hernia, stricture, etc., is preceded or attended by the local symptoms of these diseases. In like manner we judge of the nature of the diarrhœa that occurs in the course of tuberculosis or syphilis and in the course of organic heart disease or of liver disease. In each instance the possible influence of morbid processes present in other structures must be very carefully estimated.

Varieties of Acute Intestinal Catarrh. Divisions have been made in accordance with the symptoms which distinguish the various localities of the intestine in which the inflammation is most marked.

Catarrh of the Duodenum. This partakes of the nature and has the symptoms of mild gastro-intestinal catarrh, and is characterized by the occurrence of jaundice due to catarrhal inflammation of the biliary passages.

Catarrhal Enteritis. Colicky pains and rumbling are experienced. There is usually gastritis at the same time. The feces are mixed with mucus. Over the right lower quadrant there is tenderness on pressure.

Catarrhal Typhlitis. Pain in the right lower quadrant with tumor, dulness on percussion, and tenderness are present. (See Typhlitis.)

Catarrhal Colitis. The large intestine is most frequently affected. Pain and tenderness occur along the course of the bowel. The evacuations contain large amounts of mucus not intimately mixed with feces, or may consist of mucus; there is tenesmus. The association with gastro-enteroptosis and with neurasthenia must be borne in mind.

Catarrhal Proctitis. Proctitis gives rise frequently to small stools, tenesmus, pain in the left lower quadrant, with tenderness about the anus, and spasms of the sphincter. The stools contain a good deal of mucus and blood.

Cholera Infantum.

This affection occurs in children during the hot season. It is promoted by unfavorable hygienic surroundings, and is due to infected milk or food. At first there is catarrhal diarrhœa. This may continue for twenty-four hours, then vomiting and diarrhœa ensue. The stools are liquid and large in amount. At first they may contain milk-curds. The vomiting is excited by anything taken into the mouth, or by odors, or by movement of the little patient. The watery discharges are almost constant. They may be preceded by greenish or yellowish-green stools for twenty-four hours. The stools are acid in reaction, and their odor is sour. At first there is colicky pain; but when the watery discharges begin, there is only a little tenesmus. The stools irritate the skin and cause eczema. The rectum may become prolapsed. The abdomen is first distended with gas, but soon becomes retracted.

In a short time, twenty-four hours or even less, collapse ensues. Previous to the collapse the skin is hot and dry; the patient is restless. The thirst is intense, the mouth dry. The body-temperature is 103° to 104° F. With collapse the extremities become cold, the skin cool. The axillary temperature is lowered and the rectal temperature increased to 105° to

106° F. The restlessness continues, the fontanelles become depressed, the eyes sunken, the face pinched, the brows drawn. The urine diminishes in amount or may be suppressed altogether. Brain symptoms ensue. So-called hydrocephaloid symptoms follow—rolling of the head, strabismus, turning in of the thumbs, and, later, convulsions. Stupor followed by coma develops in the fatal cases. If the patient does not die in collapse, marasmus develops; ulceration of the cornea may take place; there are œdema and blood extravasation under the skin. The child emaciates and withers. On account of the weak heart and exhaustion pulmonary atelectasis or bronchopneumonia may occur. The age, the season, the presence of catarrh, with collapse and other symptoms, render the diagnosis easy.

Cholera Morbus.

The attack is characterized by sudden vomiting, followed in a short time by purging. The vomiting may be preceded by pain, or both may occur at the same time. At first the pain is seated in the epigastrium, subsequently above the navel. It is very severe and paroxysmal in character, compelling the patient to double up if lying in bed. A cold perspiration breaks out on the forehead, the extremities become cold, the face anxious, the pulse rapid. At first the patient vomits undigested food, then watery, greenish-colored fluid. The latter is bitter. Purging sets in at once or within an hour. The bowel-movements follow an attack of pain. The first passage is fecal, and may contain undigested food; the subsequent passages are watery and profuse. There are severe attacks of burning and tenesmus; the abdomen is tender around the navel and in the epigastrium. After an evacuation there is slight relief, but soon another paroxysm of pain comes on. The vomiting is excessive, and retching may be present in the intervals. Ice, or water, or anything taken into the stomach excites pain and causes vomiting. The attack subsides in twelve to twenty-four hours, and is followed by exhaustion; in rare cases collapse; and in others the attack is followed by gastrointestinal catarrh.

Cholera Nostras.

The symptoms are those of a severe gastro-enteritis, sudden vomiting, and diarrhœa. The attack usually begins in the night. The vomiting is not different from that of *cholera morbus*. The watery and brownish-colored stools become colorless and have the appearance of rice-water. Pain attends the attack, rapid prostration ensues, the extremities become cold, and collapse takes place. With the collapse there are cramps in the legs. Other muscles of the body may become cramped. The disease occurs in epidemics during the hot season, and may be mistaken for Asiatic cholera. It can be distinguished from the milder forms of cholera which precede the occurrence of the epidemic only by the absence of the comma bacillus. The bacillus of cholera nostras is found in the stools. (See Feces.)

Enterocolitis.

In enterocolitis of young children the more intense inflammation succeeds a mild intestinal catarrh. There are increased languor, great fretfulness, and fever. The early catarrh is attended by green acid stools, with lumps of casein. The tongue is furred and moist at first. It soon becomes red and dry; vomiting ensues. The stools are offensive and increase in frequency, and, in addition to the appearance first indicated, contain mucus and blood. Death may take place within the first week on account of exhaustion from the vomiting and diarrhœa.

If the disease is protracted, it is attended by great wasting, symptoms of hydrocephalus, skin eruptions, hypostatic pneumonia, and extremely feeble circulation.

Chronic Intestinal Catarrh.

The disease usually follows an acute attack, or may be chronic from the start. It may follow gastric hyperacidity and dilatation of the stomach, and is not uncommonly due to gastric achylia. It arises secondarily to portal congestion in disease of the liver and in chronic disease of the heart or of the lungs. It occurs in malaria and in the scorbutic cachexia.

The symptom is diarrhœa alternating with constipation, or diarrhœa alone. Stools may contain undigested food, or pus, mucus, and blood in small amounts. Diarrhœa may be present in the morning only under these circumstances. If the feces are examined, the eggs of parasites or infusoria may be found. The local abdominal symptoms of rumbling, flatulence, and tormina are present. There are reflex symptoms of cardiac palpitation and dyspnœa (asthma). Rush of blood to the head may occur. Often these symptoms are relieved by the passage of flatus. Chronic catarrhal gastritis usually accompanies the intestinal catarrh. The general symptoms of *anæmia*, *emaciation*, and *neurasthenia* are present. Hemorrhoids are common.

Amyloid Degeneration of the Intestines.

The symptoms are those of diarrhœa, persistent but mild in character, associated with symptoms of amyloid disease in other organs. With enlargement of the liver and spleen changes in the urine due to amyloid disease are present. The occurrence of these symptoms in a patient with syphilis, or especially in a child with bone disease or tuberculosis, points to the nature of the case.

Ulceration of the Intestines.

Duodenal Ulcer. Ulcer of the duodenum often occurs in young subjects in whom there are symptoms of chlorosis or anæmia. The causes are the same as those of gastric ulcer. It may follow boils, erysipelas, or pemphigus, and differs in one ætiological respect from ulcer of the stomach in that it occurs more frequently in the male sex. The symptoms

are obscure, and may be wanting entirely, the patient probably complaining only of intestinal indigestion. In other cases they are like those of gastric ulcer. In typical cases the symptoms are those of pain situated below the xiphoid or to the right of the median line in the region of the pylorus. The pain occurs some time after eating, and may be relieved by vomiting. There is localized tenderness on pressure. Hemorrhage may take place from the stomach, or blood be found in the stools alone. It differs from gastric ulcer only in the possible difference in location of the pain, the occurrence of intestinal indigestion and hemorrhage, and the fact that the pain comes on one to two hours after eating. It may have the same consequences as gastric ulcer—hemorrhage, perforation, subphrenic or abdominal abscess, or in rare cases stenosis of the duodenum.

Duodenal ulcer is *diagnosed* by the occurrence of melæna, which may be excessive and cause syncope and vomiting, with no blood in the vomitus; by pain, which may be in the right hypochondrium or between the navel and the right costal border; by gastralgic attacks, and by dyspepsia with constipation.

General Ulceration. Ulceration of the intestine may be due to a specific infection, and hence be symptomatic of typhoid fever, syphilis, or tuberculosis. It is nearly always present in the first-mentioned disease, and of frequent occurrence in the latter. Follicular ulceration occurs in enterocolitis in children. Ulcers due to the presence of feces occur in typhlitis and chronic constipation. The sacculi of the colon become filled with scybalous masses, the pressure of which produces ulcers. Tenderness is experienced along the course of the colon, particularly on palpation of the fecal masses, which may be felt through the abdominal wall. A *chronic ulcerative colitis* is the form that succeeds the diarrhœas which occur during camp-life, or that are set up in communities where people are crowded and live amid unhygienic surroundings. It is the form that attends scurvy, and is frequently seen in chronic Bright's disease. It may be succeeded by dilatation of the colon, by hypertrophy of the muscular walls, or by contraction of the bowel. The persistent diarrhœa leads to profound emaciation, extreme prostration, sallow complexion, with markedly impaired nutrition of the skin. Such forms of diarrhœa were seen during the War of the Rebellion, particularly in soldiers held in captivity. The diarrhœa may first be of a lenteric character, and later alternate with constipation. Stools contain blood and mucus.

Ulcers of the intestinal tract may occur from other causes, and diarrhœa may be the predominant symptom. They may be due to *cancer*; the malignant nodules may ulcerate within the lumen of the bowel. The bowel may be perforated from the exterior, on account of suppuration somewhere along its course, as in appendicitis, pancreatitis, or tuberculous peritonitis.

Symptoms. The symptoms of intestinal ulcer are usually those of diarrhœa. Ulceration, however, may be present without any symptoms whatsoever, particularly if the small intestine is affected. One or two small ulcers, on the other hand, in the lower portion of the colon, may set up continuous diarrhœa. The *stools* are composed of feces, mucus,

pus, shreds of tissue, and blood. If pus is discharged in large amount, an abscess has probably opened into the bowel. Moderate discharge of pus usually follows ulcers in the colon. Pus may be present in cancer. *Hemorrhage* is of frequent occurrence, and is an important diagnostic symptom, especially if profuse and occurring without symptoms of obstruction, of gastric ulcer, or of hemorrhoids. Fragments of tissue found in the stools may point to the nature of the process; they may be composed of the mucosa, connective tissue, or muscular coat. In dysentery the stools contain large quantities of disintegrating tissue. *Pain* occurs in many of the cases. It may be general and colicky, or circumscribed in cases of ulcer of the colon. Perforation of the intestine is followed by localized or general peritonitis. The occurrence of the latter depends largely upon the situation and the rapidity of the ulceration. When the perforation is in the posterior wall of the colon, a circumscribed abscess may develop. When it is situated in the upper zone, the pus may accumulate underneath the diaphragm or in the lesser peritoneal cavity. The signs of *pyopneumothorax subphrenicus* occur when the latter accident takes place, as both pus and air accumulate in the abscess-cavity. In such instances the ulceration usually takes place at the splenic flexure. Perforation of an ulcer of the cæcum may simulate appendicitis.

Tuberculosis of the Intestine.

The disease is usually secondary to chronic tuberculosis, but may be primary, especially in children. The symptoms are usually those of *diarrhœa*, and in the primary form this is associated with general emaciation, which advances rapidly, and with anæmia. Fever of the intermittent or remittent type is present. There is meteorism; the abdomen is much distended, but eventually becomes contracted. The mesenteric glands can be made out along the spinal column, and the intestines may become bunched into a mass, yielding a dull tympany on percussion in the centre of the abdomen. The *diarrhœa* is attended with colicky pains. The *diagnosis* is based upon the rapid emaciation, irregular fever, enlargement of the mesenteric glands in a patient, usually a child, who had probably been exposed to tuberculous infection. In one of my cases, the child, aged four years, ate of the same food, using the same utensils, as a brother, a man of twenty-two years, who was dying of pulmonary tuberculosis. The child was constantly with the brother. The remainder of the family, eight in number, remained in perfect health, and were all of good physique. The elder brother had become infected by association with tuberculous subjects in improper quarters away from home.

Intestinal Obstruction.

Ætiology. Intestinal obstruction may be acute or chronic. The causes in general may be arranged under the three following heads: diseases outside of the intestines, diseases of the intestinal walls, and accumulations within the intestines.

A. Diseases Outside of the Intestines. 1. Pressure of *tumors*, chiefly

ovarian, uterine, and omental tumors, and pelvic abscesses, or abscess about the cæcum. The obstruction may be *acute* or *chronic*. The symptoms of obstruction develop gradually, although in some instances they may take place suddenly, especially if aided by the accidental occurrence of fecal impaction.

2. *Constricting bands*, hernial openings, the remains of fetal structures, cause constriction of the intestine. In this class of cases there is usually pain, and the history preceding the obstruction is that of peritonitis, general or local, of old hernia, of appendicitis, of pyosalpinx, or of inflammation about the gall-bladder and gall-ducts. The onset may be *acute* or *chronic*. If the constriction is due to protrusion into hernial openings, the onset is usually sudden and without previous symptoms.

3. *Peritonitis* is a common cause of *acute* intestinal obstruction. It may be due to over-distention by gas and paresis of the bowel, or to pressure by external exudation.

4. Knots and twists of the intestines, usually seated about the sigmoid flexure, causing *volvulus*, are a common cause of *acute* constriction.

B. Diseases of the Intestinal Walls. 1. Invagination, or intussusception. The attack is *acute*, although the affection may continue over a long period of time.

2. Cancer and other tumors of the intestine generally lead to stricture and *chronic* obstruction.

3. The healing of ulcers, which are syphilitic in the larger number of cases, rarely tuberculous, will lead to stricture. The obstruction belongs to the *chronic* variety. It is seated, in the larger number of instances, in the rectum or sigmoid flexure of the colon.

C. Accumulations within the Intestines. 1. Feces. The obstruction takes place gradually, occurs in weak and debilitated people in the course of constipation, especially the constipation of acute disease.

2. Accumulations of improper food or foreign materials. The seeds of fruits or the husks of grain accumulate and cause obstruction. Magnesia, iron, and other articles taken as medicines, from their accumulation, may lead to obstruction of the intestine. In both of the above-mentioned varieties obstruction is *chronic*.

3. Impaction of gallstone within the intestine is followed by acute obstruction.

Causes of Acute Intestinal Obstruction. Various factors must be considered in order to estimate the cause of the obstruction.

THE AGE. Obstruction from intussusception occurs early in life; from bands or through apertures, in adult life, usually prior to forty years of age; from *volvulus*, between forty and sixty years. Obstruction due to a gallstone occurs during the middle or later period of life—always after the fortieth year.

THE PREVIOUS HISTORY. In obstruction by bands of adhesion there is a history of peritonitis, or, as Treves points out, previous attacks of obstruction more or less marked. In *volvulus* the patient has been subject to constipation prior to the attack, and in intussusception there has been no previous history, unless polypus was present, causing dragging, colicky pains, and occasional discharge of blood.

The three principal causes of acute intestinal obstruction, when it does not develop in the course of a chronic obstruction, are : constriction or strangulation of the bowel by peritoneal bands or the contraction of apertures through which the bowel passes ; volvulus of the colon ; and acute intussusception.

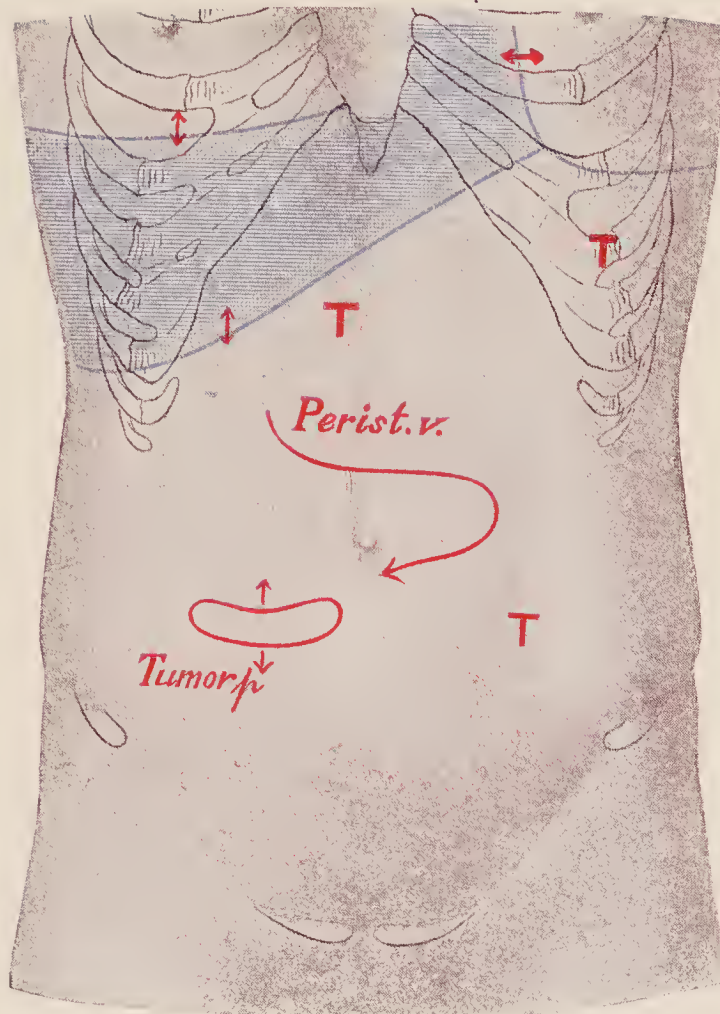
Strangulation. The type of the first variety is seen in strangulated hernia ; but similar strangulations occur in apertures within the peritoneal cavity. Thus a loop of intestine may be caught and constricted in the duodenojejunal fossa, forming a so-called *Treitz retroperitoneal hernia* ; or in the foramen of Winslow—*intersigmoid hernia* ; and, finally, there is *diaphragmatic hernia*, or protrusion through the diaphragm of the intestine along with other abdominal viscera. These irregular forms of hernia may exist without symptoms, or they may lead to constriction or twisting of the loop of intestine with the development of acute obstruction. The bowel may become caught and constricted in an omental laceration, so-called internal constriction. The commonest situations for external constrictions are the hernial regions, where the gut is apt to be constricted by the dense fibrous adhesions, and in the vicinity of the uterus and the Fallopian tubes when these structures have previously been the seat of inflammation. The constricting bands that remain after the subsidence of the local peritonitis may gradually compress the gut to the point of complete obliteration of its lumen ; or the bowel may become twisted about the adhesions with a similar result. Disease about the vermiform appendix, with secondary adhesions, has been observed to cause constriction. A common form of intestinal obstruction is caused by tangling of the intestine in the remains of the omphalomesenteric duct or Meckel's diverticulum, which is situated a short distance above the ileocaecal valve.

Volvulus. Volvulus is a rare form of acute intestinal obstruction due to twisting of the intestine, usually at the sigmoid flexure of the colon. When there is congenital elongation of this portion of the bowel, the weight of the intestinal contents, dragging upon the gut, causes it to double on itself and form a loop which then becomes twisted ; other portions of the intestine, under the influence of peristalsis, wind about the pedicle of the loop, and the obstruction becomes complete. In extreme cases true knotting of the bowel has been known to take place. Abnormal peristalsis induced by diarrhoea often precedes the appearance of the obstruction. *External injury* is said also to be occasionally responsible for intestinal obstruction.

Intussusception. (Plate XLI., Fig. 1.) Intussusception as a cause of intestinal obstruction occurs most frequently in children, and is due to invagination of the intestine, the upper portion being pushed into the lumen of the part immediately below by the active peristaltic movements, which persist in the proximal portion, while the distal portion becomes paralyzed. Intussusception is frequently found in the bodies of children who have died from exhaustion, the accident in these cases having occurred just before death. Intestinal polypi dragging one portion of the bowel down into the part immediately below are sometimes the cause of intussusception ; large portions of the bowel may thus be involved.

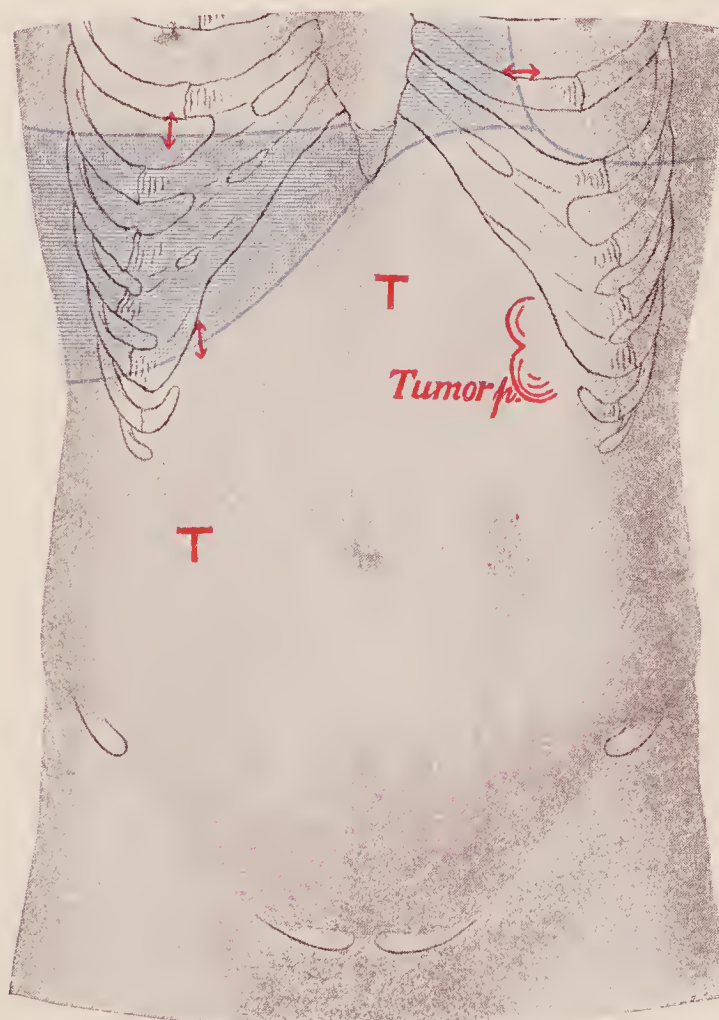
PLATE XLI.

FIG. 1.



Invagination of the Ileum.

FIG. 2.



Carcinoma of the Colon.

The invagination usually takes place in the lower portion of the ileum or in the cæcum; sometimes the invaginated loop extends to the rectum and presents at the anus. Intense inflammation, with the formation of adhesions, marks the course of the disease; the inner knuckle becomes gangrenous from the constriction of the afferent vessels, and may slough off and be discharged with feces through the anus, thus effecting a spontaneous cure.

Symptoms. When symptoms of intestinal obstruction occur, it is important to ascertain, in addition, first, the duration of the obstruction and its mode of onset; second, the possible cause of the obstruction; third, the seat of the obstruction.

Symptoms Common to all Forms of Acute Obstruction. The *local symptoms* depend upon the nature and the seat of obstruction. *Constipation*: The major symptom is stoppage of the intestinal contents. When this takes place suddenly, and there is a local injury to the bowel, the symptoms, both local and general, are severe and alarming. When constipation is complete, not even flatus escapes. *Pain*: The pain is at the seat of obstruction or about the umbilicus. It occurs suddenly, and is intense and colicky or lancinating in character, radiating from the point of obstruction. There is tenderness over the painful part. The pain is due to the injury by the constricting agent or to violent peristalsis. It may be relieved by pressure. If the pain is intermittent, the obstruction is incomplete; if constant, it is absolute. *Tumor*: In many instances a tumor can be outlined, due to single loops of intestine, thickened walls, or abnormal contents. This is particularly the case in the obstruction of invagination and the obstruction due to volvulus. *Peristalsis*: The obstruction further causes *increased peristalsis*. This takes place above the point of constriction. Sometimes the movements of the intestine can be seen through the abdominal walls. The extent of the peristalsis is an indication of the site of the obstruction. The higher the obstruction the less the peristalsis. *Meteorism*: Gas accumulates above the point of obstruction, giving rise to meteorism. If the obstruction is low down, the distention and meteorism are general. If high up, in the small intestine, from constriction by Meckel's diverticulum or internal hernia, the meteorism is in the upper part of the abdomen and may be limited in extent, or dilatation of the stomach alone may be present. *Vomiting*: Vomiting soon occurs in acute intestinal obstruction from decomposition of intestinal contents, irritation of the stomach by the intestinal contents, injury to the peritoneum at the seat of the obstruction, or, finally, to the occurrence of peritonitis. At first the contents of the stomach are ejected, then watery fluid, bile tinged or largely made up of bile, and later fecal matter. Although of fecal odor, this does not constitute true stercoraceous vomiting; the latter occurs later in the course of the disease. It must not be forgotten that any obstruction of the intestine may develop with extreme rapidity, so that *fecal vomiting* may occur within two hours of the commencement of an obstruction. It is recognized by the odor of the matter vomited and by the color. It is a grave symptom, indicating complete obstruction of the intestine. If the obstruction is high up, as in the jejunum, fecal vomiting does not

occur. Simple vomiting, however, is more persistent in high obstruction. *Eructations of gas* are frequent.

The **general symptoms** are those of *extreme prostration* or *shock* in its most pronounced form. The *abdominal facies* previously described develops very rapidly. The *tongue* is not changed at first, but soon becomes dry and brown. In a few instances, as in invagination, there may be *fever*, but in other cases usually at once, or very soon in its course, the temperature falls to or below normal, or remains at this point if it has not risen. The extremities are cold, the features pinched, the eyes sunken, the expression anxious. The pain causes the patient to double up in bed. The pulse becomes rapid, weak, and thready. The respirations are proportionately hurried, but are also made more rapid and shallow by the tympany. The mind remains clear until the supervention of peritonitis and septicæmia.

Symptoms of Various Forms of Acute Obstruction. It is essential in order to distinguish the form of acute obstruction to ascertain the *nature* of the obstruction, and to determine, if possible, its *site*. The symptoms of the various forms of acute obstruction vary somewhat. *Pain* in strangulation, from bands or hernia, is severe and paroxysmal in character and attended by collapse. It occurs early in volvulus, though it is not so severe as in the former variety, and occurs at long intervals, becoming constant with exacerbations. In acute intussusception the pain occurs early, and is steady. It increases, and then may suddenly subside. At first it is paroxysmal, attending discharge of blood and mucus from the bowels. *Local tenderness* in strangulation occurs late. In volvulus it occurs early, and may be noted over distended coils. In intussusception it usually corresponds to a sausage-shaped tumor. *Vomiting* is marked in strangulation, soon becomes feculent, and increases the severity of the paroxysms of pain. In jejunal obstruction it is excessive and non-feculent. In volvulus it does not come on so quickly, but is severe and constant when it takes place. The relaxation that attends vomiting often affords relief to the obstruction. In intussusception it does not occur as early as in the other forms, and is not so severe. It becomes feculent in only a small number of cases. *Constipation* is continuous in all cases except intussusception. In the latter there is some constipation, but it is not absolute; diarrhœa is not uncommon, and discharge of blood in the stools, according to Treves, occurs in 80 per cent. of the cases. *Prostration* is severe in all cases, although probably not so marked in volvulus. Because of its close proximity to the rectum *tenesmus* occurs in volvulus. The symptom is of frequent occurrence in intussusception, often beginning early in the attack.

Physical Signs. (Plate XLI., Figs. 1 and 2.) On palpation of the abdominal wall it is noted to be soft and flaccid in most of the cases, unless peritonitis has ensued. This occurs early in volvulus, hence rigidity is marked. A *tumor* can be made out as a rule only in intussusception. It is seated in the lower right quadrant of the abdomen. Early in the attack it is oblong and sausage-shaped. When peritonitis ensues, it disappears on account of the tympany. A portion of the gut may protrude at the anus or be felt on rectal examination. *Meteorism*

occurs about the third day in a strangulation; it occurs early, is very rapid and pronounced in volvulus; and is absent in intussusception, unless constipation or peritonitis takes place. It is not marked in high obstruction.

The Site of the Obstruction. The seat of obstruction is in a measure indicated by (1) the location of the pain or abnormal sensations, (2) the character of the swelling, (3) the character of the stools, (4) the character of the vomiting, (5) the degree of meteorism, (6) the results of a rectal examination, (7) the change in the urine, (8) the general condition. The patient is often able to indicate the location of the obstruction fairly well by the sensations of obstruction or fulness and by the great relief experienced when a free evacuation of the bowels takes place naturally or artificially. On auscultation, when the bowel is irrigated, a *murmur*, like the deglutition-murmur, may be heard at the point of constriction of the gut. In obstruction high up there is but little *meteorism*, the tumor is usually not detected, and pain is seated about the umbilicus or the upper quadrants of the abdomen. Obstruction at the ileocaecal valve may be indicated by a tumor in the lower right quadrant over the region of the valve or just above it. It is usually at this point that invagination takes place, and hence we may look for a tumor in this situation. (Plate XLI., Fig. 1.) On the other hand, in volvulus of the colon, or stricture of the rectum, the obstruction, being low down, is attended by much meteorism and by pain in the left lower quadrant of the abdomen. A tumor may be detected in this position. The position of the obstruction is sometimes indicated by the seat of *peristalsis*. This may be seen to stop at a given point, which usually indicates the position of the obstruction. The seat of obstruction may be indicated by the number of coils of intestine that are engaged in the peristaltic movement. The coils of intestine in front of the tumor are dilated and hypertrophied. In active movement they cause prominences which follow the course of the bowel. Wylie has called them "patterns of abdominal tumidity." If the obstruction is in the jejunum, peristalsis may not be observed. If the lower end of the large intestine is obstructed, the colon is prominent; if the gut about the ileocaecal valve, the region about and below the umbilicus is prominent. *The urine:* The position of the tumor, it is said, may be to some extent indicated by changes in the urine. When the obstruction is in the small intestine, indican is much increased from the decomposition of albuminous substances and products of putrefaction. In this location the urine may be suppressed. In stenosis of the large intestine indican is often not increased unless there is cancer. The value of the information derived from the character of the stools and the results of rectal examination is obvious. Obstruction in the duodenum or jejunum is followed by rapid collapse and anuria. In general, it may be said the more severe and rapid the symptoms the more likelihood that the obstruction is in the small intestine.

Strangulation. Obstruction from hernia or constricting bands occurs in adults of both sexes after the fortieth year. In stricture from pressure of bands there has usually been a history of previous attacks of peritonitis

or of inflammation of the structures in relation to the peritoneum. Hence, a cholecystitis or appendicitis is often found to precede the obstruction. The attacks begin suddenly, and the symptoms may from the start be most pronounced. They are the typical symptoms of intestinal obstruction. The local tenderness, however, may not be present as early as in other forms of obstruction. It is quite characteristic not to find a tumor or positive local cause for the obstruction, and also not to have meteorismus. This is due to the fact that the obstruction is usually high up in the intestinal tract.

Volvulus. Volvulus occurs most frequently in males. It occurs late in life, and is usually preceded by a history of constipation. Premonitory symptoms may have been present for a few days, but the symptoms of obstruction develop suddenly. They are the symptoms of acute obstruction, but as the lesion is in the lower portion of the bowel, meteorism is present to a marked degree, and rectal symptoms are found. Tenesmus is present in a small proportion of cases. Peritonitis is likely to set in early, with increase in the temperature, increased tenderness of the abdomen, and more pronounced symptoms of collapse.

Intussusception.

Intussusception (Plate XLI., Fig. 1), or invagination, occurs most frequently in children prior to the tenth year. It is characterized by severe colic and pain in the abdomen, first complained of about the navel. The severity increases in paroxysms, and only lessens if complete strangulation has taken place. With the onset of the pain there are one or two movements of the bowels, which contain mucus and blood. After this there may be constipation, or the stools continue to be loose, and are as frequent as fifteen or twenty in a day. Sometimes they are quite bloody, and almost always there is some tenesmus. In a short time after the attack vomiting commences; it may be constant or occur only after taking food. At first the abdomen is soft, but tender on pressure. A sausage-like tumor can be felt on the right side below the transverse umbilical line. On inspection of the rectum a portion of the intestine may be seen, dark and gangrenous in appearance, or it may be felt by palpation. If there is much tenesmus, the anus often remains open. In rare cases the bowel may slip back and the symptoms subside spontaneously. On the other hand, peritonitis may rapidly ensue with high fever, followed by collapse and death.

Differential Diagnosis. Intussusception must be distinguished from *enterocolitis* of childhood or the proctitis due to a polypus. In enterocolitis there is no tumor, and the collapse and prostration do not occur so early and are not so rapid. There is greater likelihood of a number of the stools being greenish like spinach. In a polypus of the rectum the symptoms are local. The child is worn out and restless; but great abdominal tenderness, and the tumor, meteorism, vomiting, and collapse are absent. The rectum must be examined.

In *peritonitis* symptoms of stenosis of the bowel from *ileus paralyticus* may be present. The history and sequence of events must be watched

carefully. Often the commencement of the affection about hollow viscera which have previously been the seat of disease, or its onset with sudden perforation will point to the nature of the affection. In peritonitis there is no active peristalsis; there is general distention of the abdomen with general tenderness; the urine is diminished, but does not contain indican in excess, except in purulent peritonitis when the amount is large. Collapse ensues rapidly. Signs of effusion within the abdomen may appear.

In *embolism* or *thrombosis* of the *mesenteric artery* and *infarction* of the bowel the symptoms take place suddenly. The patients have reached middle or late life, and have atheroma of the general arterial system. Sudden pain in the abdomen, with vomiting and symptoms of collapse, takes place. There is moderate obstruction with distention of the abdomen. After the pain diarrhoea with the passage of blood develops. The age and the absence of tumor are distinguishing features from intussusception, the only intestinal condition for which it may be mistaken.

Diagnosis of Acute Intestinal Obstruction from Other Conditions.

Acute intestinal obstruction must be distinguished from peritonitis and appendicitis. This is sometimes very difficult. Careful attention must be paid to the evolution of the case and the history of previous abdominal disease, or of lesions on account of which, on the one hand, peritonitis may occur, or on the other, obstruction of the bowel.

In *peritonitis* the attack follows disease in the uterine appendages, the vermiform appendix, or the gall-bladder, or perforation in some portion of the gastro-intestinal tract. *Fever* usually attends the inflammation, with or without chill. *Vomiting* will probably occur at the onset, and then subside until the peritonitis becomes general. The first paroxysms of vomiting are apparently due to shock. The vomiting that occurs rarely becomes feculent. As the peritonitis advances the vomiting becomes passive; a simple constant regurgitation of a large amount of fluid, greenish or grayish yellow, or watery, takes place. It pours into the mouth, and is simply discharged without the occurrence of retching. The abdomen is swollen and *tympanic*. The symptoms due to excessive tympany are more marked than in intestinal obstruction. As the movement of the diaphragm is interfered with, breathing is hurried. The abdomen is tender on pressure and is the seat of general *pain*. The general pain and tenderness, however, can usually be found to be more marked at the possible primary focus of the disease. Further, on local examination, fulness or undue prominence or *swelling* may be observed in these situations. On *palpation* over the point of origin there may be localized *œdema*. The symptoms of collapse do not differ materially from those of intestinal obstruction, although the peculiar appearance of the face and other nervous features occur more rapidly in peritonitis than in obstruction. It must be remembered that peritonitis in a large majority of cases attends obstruction.

In *appendicitis* the symptoms are somewhat like those of intestinal obstruction. There may be constipation and vomiting. The former is not pronounced, and can usually be relieved. Vomiting subsides after the first twenty-four hours, unless peritonitis supervenes; it is never stercoraceous. The local physical signs are characteristic. In appendicitis there is fixed tenderness on pressure at McBurney's point. Some

swelling can almost always be observed. On light or deep percussion there is a change in the note as compared with the other side. Fluctuation can often be detected in from two to four or five days. Both the tumor and fluctuation can be detected by bimanual examination of the abdomen and flank. Examination by the rectum may reveal a tumor at the brim of the pelvis in the right side. Fever attends the attack throughout. When peritonitis supervenes, there is rigidity of the entire abdomen, which at first was localized to the right lower quadrant.

Intestinal obstruction must not be confounded with *enteritis*. In all forms there is diarrhœa, in many vomiting. Pain of a colicky nature, spreading from the neighborhood of the umbilicus, is marked whenever obstruction to the passage of feces or gas takes place. Vomiting is not stercoraceous, and the general symptoms, collapse, etc., do not occur.

Acute hemorrhagic pancreatitis is also attended by symptoms similar to those of intestinal obstruction. There is sudden severe pain in the upper half of the abdomen, with vomiting and the rapid development of collapse; there may be constipation; the situation of the pain is of some significance. Vomiting never becomes stercoraceous; flatus can usually be passed and the bowels opened by an enema. Meteorism does not take place, although the epigastrium is tympanitic. If the symptoms are not so severe, there may be increased dullness, and possibly a tumor on deep palpation in the left upper quadrant of the abdomen along the margins of the ribs, which should be dull on percussion, or, on account of its relation to the stomach, give a dull tympanitic note. The symptoms of internal hemorrhage are present: pallor of the face and extremities, syncope, and, in addition, prostration and other symptoms of collapse.

Symptoms of Chronic Obstruction. The general symptoms are those of *chronic constipation*, with local symptoms due to the cause of the obstruction. The bowels are moved infrequently, and the stools are small. In obstruction due to stricture from cancer or cicatricial closure, the feces are ribbon-shaped. Reference must again be made to the occurrence of so-called spurious diarrhœa, with or without the passage of small scybalous masses, on account of impaction of feces. Some credence can be given to the oft-repeated expression of the patients that they have a sense of obstruction in the bowel and that they experience great relief when there is a free evacuation. In chronic obstruction the general symptoms are those of inanition, with the nervous train of symptoms that have been described in constipation; while the local symptoms depend upon the cause. When the local symptoms are due to the pressure of a tumor, or accumulation of pus or fluid within the abdomen, there is a history of local disease, on account of which the tumor developed; such history is obtained in fibroids or ovarian tumor, or in previous inflammation, which was followed by the occurrence of a tumor about the locality of the inflammation, as the pelvis or the appendix.

Chronic intestinal obstruction always occurs in *adults*. The *onset* is gradual. The *pain* that attends obstruction of this form is intermittent, and if there is fecal accumulation, it is not very prominent. *Vomiting* occurs late in the disease, is small in amount, and generally is not a

prominent feature. Obstruction to the passage of feces may be constant, or alternate with *diarrhœa*. In fecal accumulation it becomes complete, although spurious diarrhœa may attend it. The discharges may be bloody, suggesting cancer. *Tenesmus* is present in stricture low down in the large bowel. *Meteorism* is not marked when the obstruction is high up, as in acute obstruction. When the obstruction is in the large intestine, it may be extreme, and in fecal obstruction gradually increases as the obstruction becomes more marked. Coils of intestine in peristaltic movement are seen only in cases in which there is marked emaciation.

If the obstruction is due to *stricture* from *cancer* of the intestine, the symptoms of that affection are present. A tumor can be made out at some situation in the course of the bowel, usually about the cæcum or at one of the flexures. The symptoms are (1) the cachexia, emaciation, and anæmia; (2) pain; (3) tumor; (4) constipation with scybalous discharges; (5) bloody discharge; (6) mucous discharge. It is a striking fact that in many cases the symptoms of moderate obstruction come on suddenly in the midst of fair or good health. If the cancer is seated in the rectum, we find tormina and tenesmus, and the discharge of blood and scybalous masses. Local examination reveals the presence of a malignant mass. Obstruction due to *stricture* from the healing of an *ulcer* is seated in the rectum or sigmoid flexure of the colon. Pain and a sense of obstruction are referred to that locality. A history of syphilis may be obtained, and frequently the rectal tube or the finger will detect the stricture. In both there is a history of imperfect, irregular action of the bowels from time to time, with intervals of comparative comfort. These symptoms precede the constipation. When feces accumulate in the colon, the larger accumulations take place in the sigmoid flexure and in the cæcum. Fecal tumors, described under Constipation, are felt through the abdominal walls. Obstruction from *fecal accumulation* is preceded by a history of constipation (*q. v.*). The accumulations can be easily discerned as a rule. It must not be forgotten that chronic intestinal obstruction may at any time become acute.

Cancer of the Intestines.

Obstruction must not be confounded with carcinoma of the intestines. (Plate XLI., Fig. 2.) The disease usually occurs late in life, and is associated with progressive *emaciation* and *cachexia*. There may not be any symptoms save general failure of health until the sudden occurrence of obstruction of the bowel. The symptoms vary with the position of the carcinoma and the direction of its growth. The most common situations of cancer of the bowel are the cæcum, the flexures of the colon, and the rectum. In some instances with the general symptoms, there may be irregular *pain* in the abdomen, with irregularity of stools. The *tumor* may be detected if the small intestine is involved. Its detection is facilitated by having the patient get on the hands and knees, and palpating the abdomen in this position, and by clearing out the colon with a large enema. On auscultation the water may be heard to enter the dilated colon beyond the tumor, the sound resembling the deglutition-murmur

heard over the cardiac end of the stomach. If the tumor is situated in the lower colon, pain in the sacral region, resembling sciatica, may be complained of; if the cæcum or the sigmoid flexure is the seat of disease, a tumor is usually detected. Wherever the situation, the tumor found is tender, usually lying in the axis of the intestine—movable if in the small intestine, fixed if in the cæcum or the sigmoid flexure. In the latter location the tumor may be felt per rectum. One notable characteristic is that it may be palpable some days and not be present at other times. The position and size may vary from day to day, although the tumor is always hard and knotty, not doughy. By means of the proctoscope, with the patient in the knee-chest position, as described by Kelly, the presence of tumors of the descending colon will be revealed. *Constipation* is characteristic of most of the cases. It may alternate with diarrhœa. Paralysis of the sphincter ani with incontinence may take place. The stools are frequently ribbon-shaped, or they may be passed in scybalous masses; and large or oftener small amounts of blood, chiefly the latter, are passed with pus or mucus; sometimes masses resembling cancer can be found in the stools. If the tumor is in the rectum, there is great difficulty in defecation; the act is attended by pain. Later the pain becomes constant, and may radiate to the hip or the genitalia. Sometimes this pain is the only symptom complained of.

The diagnostic symptoms are: (1) the general symptoms of cancer; (2) the tumor; (3) the occurrence of constipation, which leads to complete obstruction, or obstipation, alternating with diarrhœa. Blood in the stools, with alteration in the shape of the feces, is significant.

Boas insists upon the importance of sudden occurrence of moderate obstruction in fair health in persons beyond thirty or thirty-five, and considers the observation of peristaltic waves, particularly sudden, lightning-like contractions, very important.

Enteroptosis.

It is by inspection, palpation, percussion, and auscultation that we discover the anatomical cause for the symptom-group about to be described. Attention to this affection may, however, be called only by the subjective symptoms.

This disease or physical condition, called sometimes Glénard's disease, after the physician who first called attention to its existence in 1885, is characterized by the falling down or descent of a number of the abdominal organs. This occurs on account of relaxation of the supporting ligaments, the number of which Glénard puts at six. This relaxation is largely due to a flabbiness and hence lack of support of the abdominal wall; or to strain from undue physical exertion; or to the abuse of cathartics; or possibly to injury. It is far more common in females who have borne children. It may be the result of feeble muscle-tone, following prolonged illness, and in many cases seems to be due to a congenital abnormality of the tissues, whereby the ligaments readily become relaxed. There seem to be two fairly distinct forms: one, the result chiefly of mechanical strains, etc., occurs mostly in multiparous women; the other,

depending upon the body-construction, is seen often in young unmarried women and not infrequently in men. The degree of descent, and hence the severity of the symptoms, may vary from slight displacement of one or two organs to that of the large intestine, the stomach, the liver, the spleen, and the right kidney (sometimes both). In moderate cases but two of the ligaments are relaxed—the *ligamentum colicohepaticum* and the *ligamentum gastrocolicum*; in the more severe all are affected.

Symptoms. The *objective* symptoms are due to the slight displacement, and are either purely physical or arise from the alteration of the function of the stomach and the intestines.

The earliest objective symptoms are: (1) pulsation of the abdominal aorta; (2) a linear tumor or band about midway between the xiphoid cartilage and the umbilicus, extending transversely from four to six inches in length; (3) gastropptosis, or descent of the stomach; (4) movable right kidney. Later, the liver may fall from one to four inches, the spleen become palpable, and the left kidney movable. The transverse tumor above mentioned was held by Glénard to be the thickened transverse colon. Ewald, however, seems to have demonstrated that it is the pancreas. The displacement of the viscera is recognized by the methods previously detailed for physical examination of the various organs. The patient must always be examined in the erect as well as in the recumbent position. Care must be taken to distinguish gastric dilatation from gastric descent. This can be done by careful percussion after inflation with air, by gastric diaphany, by measurement with a sound, and with fluids. Glénard laid much stress upon the *splashing sound*. This may or may not be present; it may be of gastric or intestinal origin, usually the former. It does not depend upon the displacement so much as upon the occurrence of gastric dilatation. It occurs in other affections. Stiller has shown that the tip of the tenth rib is usually freely movable in the congenital type of cases.

An objective sign of diagnostic value, to which attention has been called by Treves, is the relief the patient experiences when the lower half of the abdomen is supported by a belt or by the hands of the patient or surgeon. (Glénard's belt test.)

The objective signs of gastric origin depend upon functional or organic disease of that organ. We may have, on the one hand, only the perverted gastric secretion and digestion that go with gastric neuroses; on the other hand, we may have the perverted gastric secretion of gastritis, gastric atrophy, or dilatation, and the evidences of diminished digestive, motor, and absorptive power of these affections.

The *subjective* symptoms are due to the same cause. The displacement gives rise to local symptoms of *weight*, *heaviness*, and abdominal distress, amounting in some instances to *pain*, especially when in the upright position, and to protracted and pronounced *neurasthenia*. Later, we have the subjective symptoms of *dyspepsia*, *gastritis*, *gastric dilatation*, and *intestinal atony*, while the neurasthenic symptoms grow more aggravated.

The subjective symptoms also depend upon the functional or organic changes in the stomach and intestines, upon the displacement of the

organs, with or without the above, or upon the associated physical muscular condition of the individual and the state of the nervous system.

Glénard divided the progress of the subjective symptoms into three periods :

In the *first* there is gastric atony, when the patient experiences weight and burning after eating ; a short period of wakefulness about 2 o'clock in the morning ; a loose stool on rising ; loss of strength.

In the *second* period the patient can not digest fats and starches, and the subjective symptoms arise late in the period of digestion. A dragging sensation or a feeling of emptiness occurs about three hours after meals. The patient awakens at 2 o'clock in the morning, and remains awake for two or three hours. Constipation, at times alternating with diarrhoea, is present. There is continued loss of strength, and a tired feeling is complained of on rising.

In the *third* period the symptoms of neurasthenia are most pronounced. The patient is emaciated, and complains of a constant weight and of cramps in the stomach. Constipation is obstinate, and the stools are scybalous and mucous. The patient is much prostrated and suffers from sleeplessness. The constipation and the intestinal distress are aggravated by aperients. Enemata must be resorted to for the relief of symptoms. Intestinal catarrhal or membranous enteritis is very likely to follow.

Pain throughout the abdomen, especially when walking about or in the erect posture, is frequently complained of. Some authorities speak of tenderness on pressure over the solar plexus and of tender points along the vertebræ.

The disease is sometimes overlooked and the symptoms are attributed to neurasthenia. It is often difficult to estimate which of the two preponderates.

DISEASES OF THE RECTUM.

The consideration of rectal lesions belongs to the surgeon. It is proper, however, to insist upon the very frequent deleterious effect of such lesions in neurasthenic subjects. Indeed, the bleeding which attends hemorrhoids may be sufficient to lead to profound anæmia upon which neurasthenia may readily develop. The local suffering due to rectal fissure, or prolapse, may aggravate any tendency to the state of neurasthenia, or aid materially, with other conditions, to fasten it more firmly upon the system. In cases of anæmia, of neurasthenia, of the gastric neuroses, of debility, or prostration, the cause of which can not be ascertained, the rectum should be examined. The appearance of hemorrhoids and other rectal affections is described in works on surgery. Hemorrhoids, ulcers, fistula, and carcinoma are to be sought for in abdominal affections.

Inspection and palpation are necessary. The symptoms are those of local pain, tenesmus, and frequently hemorrhage. The pain follows a movement of the bowels. There may be a feeling as of a foreign body in the rectum, with some itching and burning about the anus. The pain may be so severe as to inhibit defecation. Timid subjects will not endure the act ; in consequence they suffer from vertigo, headache, tym-

panites, and symptoms of gastro-intestinal disorder. In some instances there is chronic catarrh of the rectum, with discharge of small stools containing mucus or pus streaked with blood. Cases occur in which hemorrhage is the only symptom, the constant recurrence of which leads to grave constitutional results. Hemorrhoids are the lesions for which the rectum is most frequently examined. They, as well as other lesions, are of diagnostic significance in affections beyond the rectum. Thus in all forms of portal congestion internal hemorrhoids are of constant occurrence, and when found in a toper may be one of the first indications of cirrhosis of the liver. Rectal fissure is not of much diagnostic significance. The finding of a small cancer, the symptoms of which may be those of hemorrhoids, may explain emaciation and the development of cachexia. Ulcer of the rectum may be due to syphilis, cancer, or tuberculosis. A fistula is often tuberculous. The rectum must be examined in cases of pyæmia, particularly of the portal variety, when jaundice, enlargement of the liver, and hectic fever are present, for local rectal disease may cause pylephlebitis.

CHAPTER X.

DISEASES OF THE LIVER, SPLEEN, AND PANCREAS.

THE LIVER.

THE symptoms of disease of the liver are due to the morbid processes, to disturbance of the functions of the hepatic cells, or to obstruction of the channels for the flow of blood and bile. As these latter extend beyond the glandular structure of the liver, obstruction of channels and physical alteration in the size and shape of the organ may be caused by disease outside of the organ. Hepatic symptoms may, therefore, be due to diseases other than those of the liver.

The *morbid processes* are the congestions, the inflammations, the degenerations, the morbid growths, and gross parasites.

Symptoms due to the Morbid Process. In *congestion* of the liver the symptoms are (1) the symptoms of the basal lesion, (2) enlargement of the organ from the increased amount of blood, (3) functional disturbance from the same cause. The congestion is not limited to the vessels in relation with the liver-cells, but involves the vessels of the mucous membrane also, hence the latter swell, obstruct the ducts, and produce a moderate degree of jaundice. The *inflammations* are toxic and infectious. The symptoms are due to the primary intoxication or infection, to the degree of obstruction of the vessels and ducts, and to the alteration of the shape and size of the liver and of its function. When the inflammation is diffuse, as in the cirrhoses, the hepatic symptoms are more marked; when localized, as in abscess, the infectious symptoms predominate. When the ducts are the seat of infection, the bile-channels are obstructed and jaundice ensues, while obstruction of the vessels produces ascites. In *morbid growths* of the liver the symptoms are those of malignant disease in general, to which are added symptoms due to the change in the size of the liver, and, more frequently than in inflammation, symptoms due to obstruction of the channels. The *degenerations* are so frequently secondary to and masked by the primary cause that, save in regard to change of size, there are no hepatic symptoms worth mentioning.

Symptoms due to Functional Disturbance of the Liver. The functions of the liver are to secrete bile; to destroy the hæmoglobin of the blood; to destroy, modify, or neutralize poisons, and to modify and render available for nutrition the peptones absorbed by the portal circulation; and the elaboration of glycogen. When the liver-cells are destroyed, as in acute yellow atrophy, the secretion of bile is abolished, and the liver does not destroy the usual amount of hæmoglobin. On the other hand, hæmoglobin may be so much in excess that the liver is not equal to the task of destroying it, and *non-obstructive jaundice* results. Functional

disturbance of the liver manifests itself clinically by symptoms due to the entrance into the circulation of imperfect products of digestion, or poisons which the liver has failed to destroy.

Symptoms due to Obstruction of the Channels. (1) Obstruction of the *bile-ducts*, either from disease or from external pressure, causes jaundice, pain, and fever. The three symptoms may occur singly or combined. In obstruction by *gallstones* jaundice may occur alone or be accompanied by pain; or jaundice, pain, and fever may occur together; rarely, pain or fever is present alone. Each symptom will be described later. (2) Obstruction of the *blood-channels* causes *congestion* of the liver, which may be active or passive, *portal obstruction*; here again the symptoms are modified by the process. Thus in portal obstruction from pressure the symptoms are quite different from those in portal obstruction due to suppurative inflammation of the vein.

Historical Diagnosis. Primary liver disease is comparatively rare; secondary liver disease, on the other hand, is of common occurrence. For this reason a knowledge of ætiological factors is of aid in the diagnosis of hepatic affections. There are but few general organic diseases or states of the system that do not in some way influence the liver. Functional disorders of the liver, as previously remarked, are so difficult to separate from functional disorders of the stomach and intestines, that from an ætiological and clinical standpoint they practically go hand-in-hand.

SOCIAL HISTORY. *Age.* Diseases of the liver usually occur late in life because the causes upon which they depend are operative only at that period. In a case, therefore, of ill health in a young subject, when the cause can not well be determined, the liver is not so likely to be the seat of disease as in older subjects. Late in life we have gallstones with their multiple consequences, inflammation, cirrhosis, and cancer; nevertheless, we may, although not so frequently, have the congestions and the degenerations in early life.

Sex. The sex is not of much significance from a diagnostic standpoint. Cancer may be more frequent in the female sex because cancer of the uterus and other organs is more common. Cancer of the biliary passages is more frequent in females, because in that sex gallstones, which are ætiological factors in cancer, are more common. Cirrhosis is the result of alcoholism and consequently more frequent in men.

Habits. It is always necessary to inquire into the habits. Alcoholism points to cirrhosis; the excessive use of stimulating foods to hyperæmia; sedentary habits and the use of starches and fats to gallstones. The occupation has but little influence on the development of hepatic disease. With regard to the *climate*, it may be said that in tropical countries hyperæmia and abscess of the liver are more frequent.

FAMILY HISTORY. But little is gained for the diagnosis by a study of the family history, as most of the morbid processes are secondary to disease elsewhere, except as regards biliary calculi, the formation of which appears to be very commonly a disease of special families.

PREVIOUS DISEASE. It is absolutely essential, in order to establish a diagnosis, to inquire into the previous medical history, as liver disease is

usually secondary. The occurrence of heart disease or obstructive lung disease points to congestion; infectious diseases to cirrhosis when that condition is not otherwise accounted for; dysentery to abscess; ulceration or suppuration in the portal area to multiple abscess; syphilis to syphilitic disease; tuberculosis, suppurations, bone disease, and syphilis to amyloid disease; pyæmia to multiple abscess; tuberculosis to fatty liver.

Subjective Diagnosis. Pain. Pain in biliary affections, including diseases of the passages, may be constant as in perihepatitis and carcinoma, or paroxysmal, recurring at long intervals, as in cholelithiasis. In congestion and in carcinoma it may be dull and aching, with some sense of distention. It is sharp, lancinating, increased by breathing in perihepatitis; sharp, colicky, or spasmodic in cholelithiasis. In diseases of the gall-bladder and gall-ducts it is associated with spasm of the rectus muscle, with tenderness on pressure and on special movements, as in so-called "hooking under the liver," the value of which Murphy points out in cholelithiasis. The pain of gall-bladder disease is due to inflammation, to distention, to colic or spasm induced by a foreign body (gallstones).

Pain due to inflammation and spasm must be distinguished from :

1. Dietl's crises, in floating kidneys, which may be accompanied by jaundice.
2. Crises occurring in dilated and displaced stomachs, first described by Kussmaul, due to kinking and stenosis of the first and second portions of the duodenum, especially if adhesions have taken place.
3. Crises due to downward displacement of the liver (36 per cent. of cases—Steele).
4. Renal calculi.
5. Pancreatic pain: (*a*) hemorrhagic pancreatitis; (*b*) acute pancreatitis, occasional jaundice; (*c*) pancreatic colic, crisis, and sometimes jaundice.
6. Gastric ulcer with local peritonitis.
7. Duodenal ulcer; jaundice is present in a small proportion of cases.
8. Gastric neuroses.
9. Abdominal pain of thoracic disease, as pneumonia, diaphragmatic pleurisy.
10. Disease of the vertebræ—tuberculous and rhizomelic.
11. Appendicitis.
12. Intestinal pain; (*a*) acute ileocolitis; (*b*) lead colic; (*c*) epigastric hernia.
13. Gastric crises of locomotor ataxia.
14. Peritoneal pains of toxic origin often occurring in uræmia—pain in upper half of abdomen, more or less collapse with nausea and vomiting.
15. Pain resulting from a forgotten fracture of a rib with callus pinching the nerve.
16. Aneurism of the aorta.
17. Obscure alleged neuralgias of the liver.

Objective Diagnosis. Jaundice. Jaundice presents two distinct forms—*obstructive* and *non-obstructive*, sometimes also called hepatic and extra-hepatic. The old terms hepatogenous and hæmatogenous owe their origin to the erroneous belief that bile-pigments, which are usually produced in the liver, may sometimes result directly from blood-destruction without the intervention of the liver. There is no doubt, however, that bile-pigments are always formed in the liver; hence the term hæmatogenous jaundice is incorrect.

GENERAL SYMPTOMS. The color of the skin and of the mucous membranes has been described elsewhere. (See page 206.) In addition to the yellow discoloration we find: 1. *Cutaneous irritation.* An attack of jaundice may be preceded by general itching. The pruritus, which may be sufficiently severe to cause great distress, occurs in all forms of icterus, but is most marked in obstructive jaundice of long duration. Scratch-marks are seen on the surface of the skin, and boils and erythematous eruptions frequently occur. *Xanthelasma* is a peculiar affection occurring on the tongue, the skin of the eyelids, and about the ears. (See page 173.) 2. *Discoloration of the secretions.* All the secretions of the body are changed in color, as previously described. 3. *Absence of bile from the feces.* The stools are ashy or gray in color. 4. *Retardation of the pulse.* The heart's action falls to 40 or 30 beats in the minute, or even lower. 5. *Hemorrhages.* In the later stages of all forms of jaundice hemorrhages are of common occurrence. In acute malignant jaundice they are seen underneath the skin and in mucous membranes. 6. *Cerebral symptoms.* Irritability and depression of spirits are marked. As the disease advances the mind grows sluggish, the patient is dull and sleeps most of the time; gradually the symptoms of the typhoid state develop. In the acute febrile forms coma and convulsions are of common occurrence. In the affection known as *acute yellow atrophy* the cerebral symptoms are marked and occur soon after the onset of the disease. Within the first twenty-four hours there may be convulsions, with delirium in the intervals, and subsequently coma.

Obstructive Jaundice. The obstruction may take place in the large ducts or in the smaller terminal ducts; it may be due to disease outside of the ducts, to disease of the ducts themselves, or to obstruction within the ducts.

1. **JAUNDICE FROM DISEASE OUTSIDE OF THE DUCTS.** *External pressure* by tumors of the stomach, kidney, pancreas, or omentum; by tumors of the liver itself or enlarged glands in the fissure of the liver; by accumulated feces in the colon; by an abdominal aneurism; and finally by the pregnant uterus, may cause obstructive jaundice. In the large majority of cases this form of jaundice is due to *disease of the pancreas*, particularly carcinoma. The condition increases gradually in severity, varying with the degree of pressure, and becomes chronic, except when due to pregnancy or fecal accumulation; it may cause death or persist until such termination results from the primary disease. It is recognized by the absence of pain; the presence of disease in other localities, indicated by its peculiar symptoms and signs; the absence of a history of gallstones; and, finally, by the patient's age. Its

nature must be inferred from the symptoms and physical signs of disease in neighboring structures. Thus, enlargement of the lymphatic glands may be inferred from the presence of primary carcinoma in other organs of the body, or from the condition of the lymphatic glands in other parts. If they are the seat of malignant disease, it can usually be recognized. Cancer of the liver must be excluded by the absence of the characteristic symptoms—enlargement with jaundice, moderate fever, rapid emaciation, and short duration of the disease.

2. JAUNDICE FROM DISEASE OF THE DUCTS. Catarrhal, suppurative, and adhesive inflammation, and cancer or other tumors of the ducts cause jaundice.

The most common form is that due to *catarrhal inflammation* of the ducts. The jaundice comes on suddenly, at least within forty-eight hours after the onset of the symptoms; there is no pain, but the attack is attended by vomiting and other symptoms of mild gastritis, and is usually accompanied by itching. It follows indiscretions in diet, and occurs in young subjects. A definite cause for the gastritis can usually be found. The *diagnosis* is based upon the age; the association of the jaundice with gastritis, for which a definite cause can often be assigned; the absence of organic heart disease, or any lesion within the body that might be responsible for the jaundice; the moderate degree of jaundice; the absence of emaciation and symptoms of portal obstruction; and the presence of moderate enlargement without pain. It must not be forgotten that jaundice due to obstruction from gallstones, or to pressure from tumors outside of the duct, is characterized in its onset by symptoms similar to those just mentioned. It is often necessary to wait before giving an opinion; when there is a history of previous attacks of jaundice in a patient more than forty years of age, great caution should be exercised in giving a diagnosis.

When the jaundice is due to *suppurative inflammation of the ducts*—suppurative cholangitis—the infection is usually associated with a previous history of gallstones. It must not be forgotten, however, that other causes of jaundice, such as obstruction by external pressure, may invite an infectious inflammation of the ducts also. The jaundice runs a chronic course and is attended with fever and other symptoms of an infection. In *adhesive inflammation* there is a history of trauma from gallstones, and the infection is chronic. In *cancer of the gall-ducts* the advent of jaundice is slow, the course protracted; the symptoms are the symptoms of carcinoma, to which are often added the physical signs of an enlarged gall-bladder. (See Diseases of the Gall-ducts.)

3. JAUNDICE FROM OBSTRUCTION WITHIN THE DUCTS. Foreign bodies within the ducts—inspissated mucus, gallstones, and parasites, such as round worms or hydatid cysts—are the common causes of occlusion of the ducts with resulting jaundice.

Foreign bodies within the ducts cause jaundice either by direct obstruction or by the catarrhal inflammation which their presence excites. The symptoms occur suddenly in the former instance, gradually in the latter. The characteristic symptoms of *gallstones* precede the jaundice. The patient is usually a woman past forty years, with habits of life that pre-

dispose to the formation of calculi. Colicky pains occurring in paroxysms, intermittent jaundice varying in intensity, and an intermittent fever point to this form of obstruction.

Jaundice due to *lowering of the blood-pressure* in the liver, so that the tension between the bile-ducts and the blood-passages is altered, occurs suddenly, is light in degree, and is not attended by marked symptoms; it is due usually to shock or emotion.

Non-obstructive or Extrahepatic Jaundice. Jaundice is *non-obstructive* when (1) the function of the liver-cells has been suppressed, as in acute yellow atrophy of the liver; (2) when blood-destruction is in excess of the capacity of the liver to remove the product of destruction—the urobilin, as in certain forms of malaria, in pernicious anæmia, in certain fevers, and in other toxæmias. The onset of the jaundice is rapid, the general symptoms are more pronounced, particularly the cerebral symptoms. They occur simultaneously with the yellow discoloration. They are *infectious*, as in acute yellow atrophy of the liver and in Weil's disease. The *toxic* forms of non-obstructive jaundice are not severe; the skin is light yellow, and the discoloration may not even be observed by the patient, or cause pronounced symptoms. The blood is destroyed rapidly in these cases; and as it can not be disposed of by the liver, spleen, or kidneys, the transformed hæmoglobin is deposited in the tissues. In this class of cases the urine contains but little bile-pigment, but there is a large amount of urobilin and indican. The stools are not clay-colored.

Malignant or Infectious Jaundice—Acute Yellow Atrophy of the Liver. Acute diffuse inflammation of the liver with necrosis of the cells, characterized by jaundice and cholæmia, is most common prior to the thirtieth year and is said sometimes to follow fright. Many of the cases occur during pregnancy. The symptoms are local and general. The discoloration is first noticed after an attack of gastroduodenal catarrh, it occasionally extends over the entire body, and, as a rule, there is only moderate itching. After a continuance of these mild symptoms for from two days to two weeks the patient complains of headache, and delirium sets in with stupor and convulsions. The headache is attended with vomiting. Fever of moderate degree begins at the same time, although in some cases it is absent.

Although the jaundice is not intense, the effects upon the blood are early seen; hemorrhages underneath the skin and from the mucous membrane take place. In pregnant women abortion results, the hemorrhage following which may be excessive. The stupor and delirium are followed by coma, and death takes place in the first week; or coma may be preceded by the typhoid state, and the disease last longer than a week. The urine is bile-stained, and contains albumin and casts. It is diminished in amount, and is soon passed involuntarily. Leucin and tyrosin are always present. The latter may be seen in the sediment, although it is more marked when a few drops of urine are evaporated on a cover-glass. The bowels are loose and the clay-colored feces are passed involuntarily.

On examination the liver is found diminished in size; this may not be

appreciated by percussion in the anterior region, but in the axillary region the width is reduced by 1 to 2 inches. There may be some tenderness over the liver and over the ducts.

DIAGNOSIS. The data upon which a diagnosis is based are the age, sex, pregnancy, the rapidity of onset of cerebral symptoms following jaundice, diminution in the size of the liver, and the finding of leucin and tyrosin in the urine. Acute yellow atrophy must be distinguished from the jaundice of hypertrophic cirrhosis of the liver, which at times becomes malignant. Some observers have thought that acute yellow atrophy may supervene upon this form of cirrhosis, thereby causing malignant jaundice; but there is more fever than in atrophy, while leucin and tyrosin are not found in the urine. It must not be forgotten that all cases of jaundice may terminate suddenly with delirium, followed by coma, or in the development of the typhoid state.

In *phosphorus-poisoning* the hemorrhages, the jaundice, and diminution in the size of the liver are the same as in acute yellow atrophy. Gastric symptoms are more marked, and there is more distinct swelling of the liver in the early stages, which is also more persistent.

Weil's Disease. This infection, in which *jaundice* is the chief symptom, has been considered in the chapter on Infectious Diseases.

Yellow Fever. The account of the jaundice attending this infection is found in the chapter on Infectious Diseases.

Infantile Jaundice. Jaundice in infants is due to two causes: *congenital obliteration* of the ducts and *catarrhal inflammation*. It must not be confounded with the yellow discoloration of the skin due to an excess of coloring-matter in the blood which is not disposed of by the liver; or perhaps to patency of the ductus venosus, as suggested by Quinke.

In *congenital obliteration* of the gall-ducts jaundice rapidly ensues and deepens to an intense degree; hemorrhages occur, the child becomes stupid or comatose, and may have convulsions; death takes place in coma. There is rapid emaciation, and the liver and spleen are enlarged. In some cases life is prolonged for many months.

Simple *catarrhal jaundice* in infants is associated with moderate gastric disturbance. The jaundice is light; the conjunctivæ alone may be discolored. In infants malignant or infectious jaundice may be due to inflammation of the portal veins secondary to umbilical phlebitis. The jaundice develops after suppurative inflammation about the umbilicus, and is attended by fever. There may be some tenderness over the liver; frequently peritonitis develops at the same time. Pyæmic symptoms may set in, and pus may be found in other situations. If death does not ensue early, the jaundice becomes more pronounced and causes cutaneous and mucous hemorrhages. Convulsions and coma are apt to supervene before death. Jaundice in infants also occurs in *interstitial hepatitis of syphilitic origin*. The evidences of hereditary syphilis are seen in the skin and mucous membranes. The liver is enlarged, and there may be tenderness from perihepatitis.

Hepatic Fever. The occurrence of fever may be of importance in distinguishing the various forms of obstructive jaundice. Fever occurs

frequently in jaundice, but is significant in certain forms only. In *catarrhal jaundice* it is present for three or four days only, disappearing as the severe gastric symptoms subside. It is probably toxic. In *hepatic colic* with jaundice it is transitory and associated with chills and sweats. In jaundice from *obstruction* it occurs with the onset of primary or secondary infectious cholangitis. A peculiar type known as *intermittent hepatic fever* (see page 366) is often seen. The intermittent fever is associated with gallstones in the following groups: First, with each paroxysm of hepatic colic moderate fever and jaundice are present. The latter becomes more intense after each paroxysm, but disappears in a short time. The paroxysmal attacks may recur at intervals for years. Second, the hepatic colic is attended by distinct ague-like paroxysms of chill, fever, and sweat, after each of which the jaundice, which continues to the end, is more intense. Third, hepatic colic and gastric disturbance occur with fever, but without jaundice. The symptoms occur in distinct paroxysms. *Gallstones* are probably the cause in all these conditions, leading in some cases to chronic obstruction of the duct without infection.

When *infectious cholangitis* with or without gallstones is present, the symptoms are somewhat different, although the fever is of the same type. Thus (1) there is more tenderness in the hepatic region, with enlargement of the gall-bladder; (2) the paroxysms are more frequent; (3) jaundice is not so intense and not paroxysmal; (4) the patient is ill in the intervals, and there is wasting. There are no periods of either local or general improvement. The most important diagnostic point in cases of gallstone is the subsidence of all symptoms between the paroxysms of fever.

Intermittent fever of this character must be distinguished from *malaria*. The history of gallstones, with pain in the region of the liver, and the negative findings in the blood, are sufficient to establish the diagnosis. This type, usually associated with ball-valve calculus, may be confounded with septicæmia (blood-cultures, streptococcus and gonococcus, or examinations of pus). Endocarditis (same means of diagnosis), with recurring fever (Ebstein's disease) due to lymphatic tuberculosis (tuberculin test). It is this type of fever that is seen in syphilis, and such a cause must be excluded. Murphy calls attention to the rapid rise and fall of the temperature in such cases.

Hepatic fever also occurs in cancer when the neoplasm grows rapidly, in certain forms of cirrhosis, and in obstruction from other causes than gallstones. It is particularly common in suppurative inflammation of hydatid cysts, or after they rupture and discharge into the biliary vessels. Without previous knowledge of the hydatid cyst the diagnosis is almost impossible, save that the pain is less when the obstruction is due to this cause than in obstruction from the passage of gallstones.

Physical Diagnosis. The diagnosis by physical means of exploration is considered in the chapter on Physical Diagnosis. It is necessary to differentiate enlargement of the liver from a false enlargement of the organ due to diseases above the diaphragm as pleural effusions from accumulations of fluid between the diaphragm and the liver; and from enlargement below the diaphragm due to fecal accumulation in the colon, renal enlargement, or tumors of adrenal origin.

Physical diagnosis of diseases of the gall-bladder and gall-ducts yield the following results by the special methods of investigation.

1. *Inspection*.—Fixation or restriction of side.

2. *Palpation*.—The presence of pain, tenderness, swelling, or spasm.

(1) Simple palpation. (a) Tenderness at Mayo-Robson's point, at junction of outer one-third with inner two-thirds of a line drawn from the tip of the ninth right cartilage to the umbilicus. (b) Outline tumor. If inflamed gall-bladder, continuous with liver; if distended, movable, pear-shaped, neck toward liver, having a tendency to disappear and bob up again.

(2) Bimanual. Fluctuation—gallstone rubbing.

(3) Method of Glénard.

(4) Eight fingers. Pressure upon four placed over part by four of other hand, the fingers of hand not pressing relaxed. (Pottalschek.)

(5) Hooking fingers under rib in region of gall-bladder or pressing deeply in this region while patient takes full breath. (Murphy.)

(6) Deep prod with closed fist (Jordan-Lloyd) over gall-bladder excites pain in cholelithiasis.

3. *Percussion*.—Outline tumor; note if (a) continuous with liver, (b) an interval of resonance which may exist between the liver and gall-bladder tumors. Auscultatory percussion not conclusive. Note, however, that percussion must be employed in both upright and recumbent postures to detect displacements of liver.

4. *Auscultation*.—Friction of perihepatitis; gallstone crepitus (rare).

X-Rays. Of some value in the hands of Beck. Results usually considered doubtful.

Laboratory Diagnosis. *The Blood—Leucocytosis.* It is remarkable how few reports are made. More precise information is required on this important point. Leucocytosis is present in acute cholecystitis and cholangitis other than typhoidal. Its presence points to streptococcus, pneumococcus, or *Bacillus coli* infection, and excludes typhoid, malarial, and tuberculous infections. It is often absent in amœbic abscess, but does not exclude multiple abscess of the liver. If lymphocytes are in excess, syphilis is suggested.

Iodophilia. Locke and Cabot found positive reaction in 5 of 7 cases of gall-ducts. Their studies, however, showed positive results in a very large percentage of cases in which suppuration was present. One can place about the same value upon iodophilia as upon leucocytosis.

Red-blood Count. This should be made repeatedly. In jaundice the number of red cells falls rapidly, and the degree of reduction should guide one in the indications for operation.

Coagulation-time. A close watch of the blood should be made with Wright's tubes. The normal coagulation-time is two to four minutes. In jaundice the blood may coagulate so slowly that eight to ten minutes elapse before clotting is completed.

Tuberculin Test. Jaundice may be due to enlarged lymphatic glands pressing on the ducts. Recurrent fever (Ebstein's) attends it. It may simulate gall-bladder infections. A tumor of the liver so situated as to resemble an enlarged gall-bladder may be tuberculous.

Congestion of the Liver.

Active Congestion. The liver is enlarged and may be tender. Weight and fulness, and often a painful sense of distention are complained of. Active hyperæmia may follow a chill or suppression of the menses ; but the commonest exciting causes are indiscretions in diet and the free use of alcohol or highly seasoned food, followed by an attack of acute gastro-intestinal catarrh. The disease is quite common in the tropics from suppression of perspiration. It is recognized by the occurrence of symptoms of acute gastritis, with enlargement, pain and tenderness of the liver, and sometimes a moderate degree of jaundice.

Passive Congestion. Passive hyperæmia also leads to enlargement of the liver which may occasion a sense of weight and fulness, but pain is not present. The organ is often not tender, although sometimes distinctly so ; the edges are smooth and indurated. Pulsation of the liver, which is sometimes present, is detected by placing the hand over the surface of the liver, when with each impulse of the heart the organ can be felt to expand. The symptoms of the cause of the passive congestion combine with those due to the enlargement, and in addition we have symptoms of obstruction to the flow of blood in the portal circuit. Pick has described what he calls "pericarditic pseudocirrhosis of the liver" in which, owing to passive congestion from pericarditis, the liver becomes cirrhotic, and ascites and other symptoms of cirrhosis predominate in the clinical picture. Since adhesive pericarditis is difficult to diagnosticate, the cases are likely to be mistaken for true cirrhosis. The condition is not very uncommon in children.

Passive congestion occurs in organic heart disease after compensation has failed and the right heart is dilated, the organ rapidly becoming congested because of its proximity to the overfilled ventricle. Mechanical congestion also takes place in emphysema of the lungs, in fibroid phthisis, and in the presence of intrathoracic tumors pressing upon the vena cava. The recognition of passive congestion is not difficult. The symptoms due to enlargement (see Objective Symptoms) and the symptoms due to portal obstruction point to the true nature of the morbid process.

Portal Congestion. Occlusion and overfilling of the branches in the liver occur in portal congestion, and most typically in cirrhosis of the liver. The circulation in the liver is interfered with ; the blood is thrown back into the portal vein and overfills the vessels of the portal area. As a result we have (1) congestion of the mucous membrane of the stomach and bowels, with the symptoms of gastro-intestinal catarrh. (2) Dilatation of the veins, chiefly the hemorrhoidal, giving rise to hemorrhoids. (3) Ascites. (4) Hemorrhages. The *hemorrhages* may occur in any part of the gastro-intestinal tract. Hæmatemesis and intestinal hemorrhage are seen separately or combined. The vomited blood may be small in amount ; often it is mixed with mucus. In some cases large and even fatal hemorrhages take place either from the mucous membrane of the stomach or from the veins about the œsophagus, which often become varicose in cirrhosis. Intestinal hemorrhages may originate in enlarged

hemorrhoidal veins, an intestinal ulcer, or the intact mucous membrane. (5) Enlargement of the spleen. (6) Changes due to the collateral circulation. If complete collateral circulation is established, the above symptoms may not ensue. The *collateral circulation* may be through deep-seated or through superficial veins. In the latter case the *external veins* of the abdomen are enlarged; the epigastric and mammary veins become prominent, and the veins about the umbilicus may become so enlarged and prominent as to form a radiating tumor to which the term *caput Medusæ* has been applied. The venules along the line of attachment of the diaphragm in the lower thoracic zone are over-distended and may be the seat of pulsation.

In consequence of the portal overfilling, the enlarged terminal branches of the vein press upon contiguous structures, interfere with the circulation of the blood in the major vascular system of the liver, and invite catarrh of the terminal ducts, with obstruction, and hence *jaundice*. This is seen quite frequently in passive congestion of the liver, rarely in cirrhosis.

Abscess of the Liver.

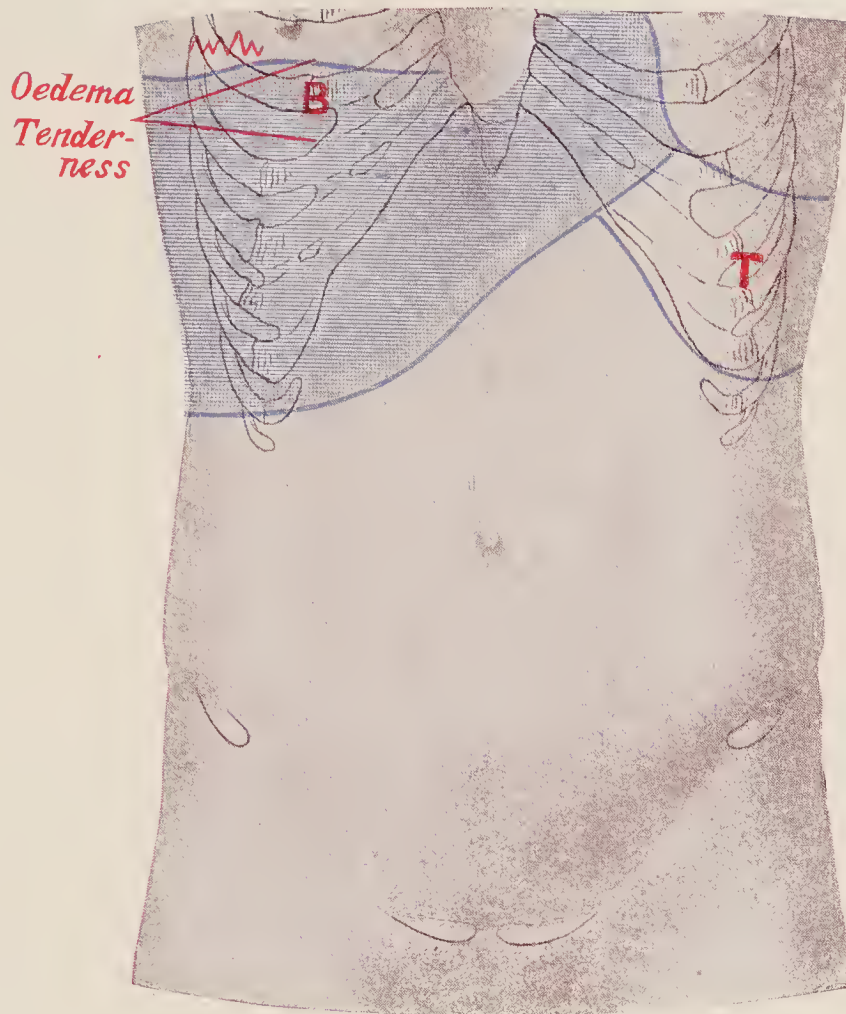
Two forms are seen: so-called *single* or *tropical abscess*, in which one or two abscesses are found; and *multiple abscesses*, found throughout the liver structure. The single or solitary abscess usually occurs in the course of dysentery, and, in all probability, in the amœbic form only; it may also be due to traumatism, particularly in children. Multiple abscesses occur secondarily to inflammation somewhere in the portal area. Inflammation and abscess about the rectum, inflammation of the appendix, ulceration anywhere in the gastro-intestinal tract may be followed by multiple hepatic abscesses. The abscesses, however, are not directly produced by emboli, as in the case of amœbic abscess, but after inflammation of the portal vein or *suppurative pylephlebitis*. Multiple abscesses of the liver also follow obstruction and infectious inflammation of the biliary passages (*suppurative cholangitis*).

Tropical or amœbic abscess varies in its clinical course. In a typical case the clinical picture is that of general suppuration developing in the course of, or soon after, an exacerbation of amœbic dysentery with local symptoms referred to the liver.

Symptoms. The *general symptoms* are those of intermittent fever, paroxysms of which may occur daily or only every second day, attended by chill, fever, and sweat. The fever may be remittent or continuous. The complexion in tropical abscess of the liver is peculiar, as all writers upon tropical diseases agree. The skin is sallow, the complexion muddy, the face pale. Through this a slightly icteroid tint may be seen, and the conjunctivæ are bile-tinged. Distinct jaundice is rare. On account of the enlargement the patient complains of weight and fulness in the region of the liver and suffers from cough and dyspnœa; vomiting, although not severe or even constant, is sometimes present. The appetite is lost and nausea at the sight of food is pronounced. The condition of the bowels varies with the state of the intestinal tract at the time of the hepatic complication. The dysenteric symptoms may subside entirely or

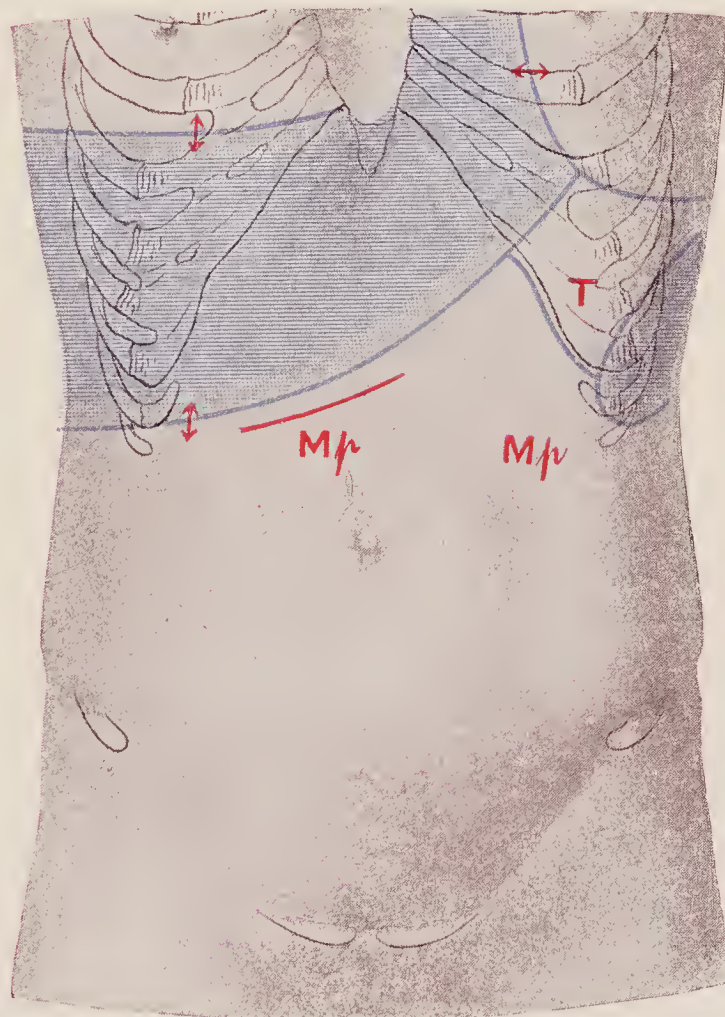
PLATE XLII.

FIG. 1.



Abscess of the Liver.

FIG. 2.



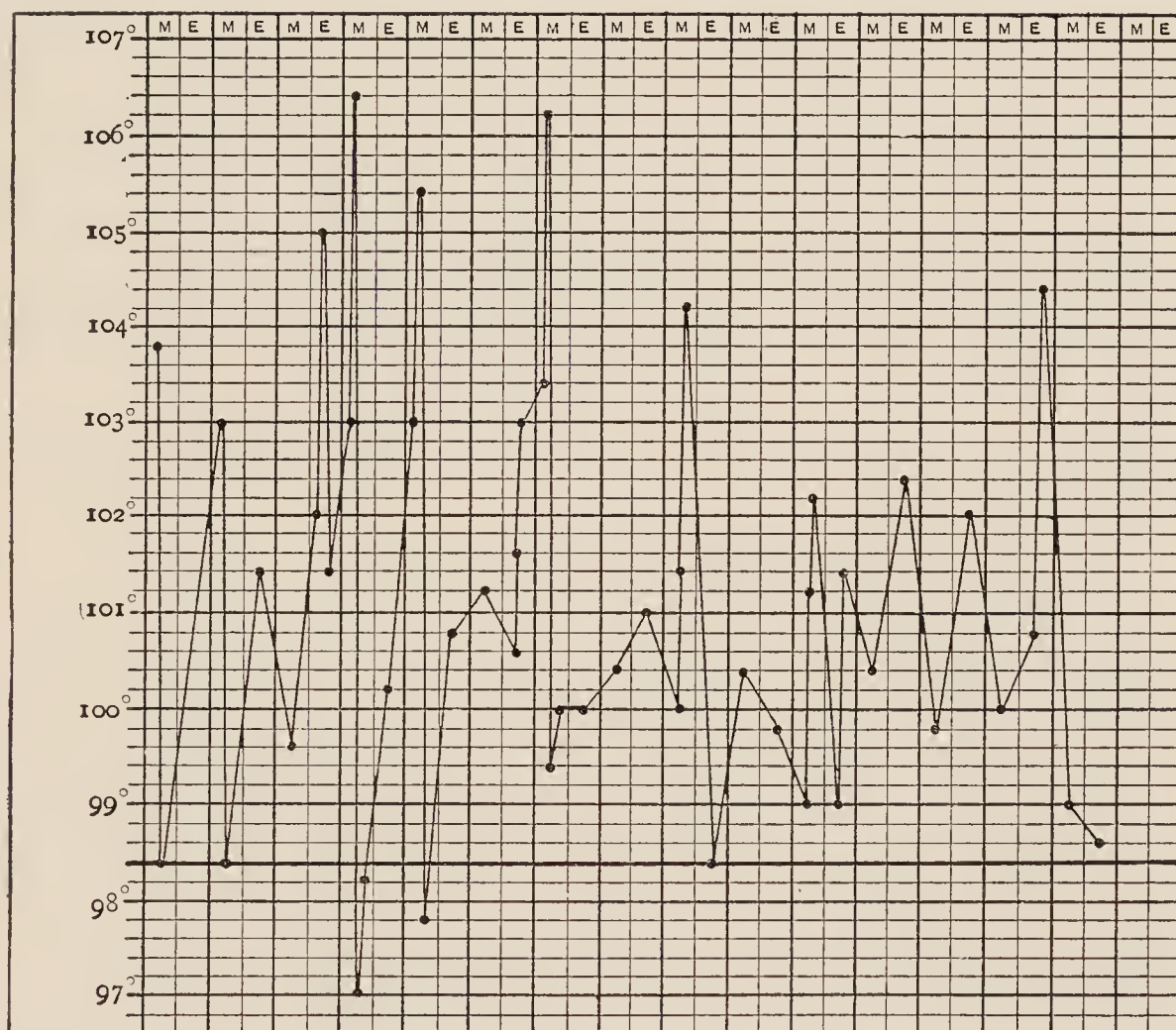
Hypertrophic Cirrhosis of the Liver with Enlargement of the Spleen.

they may continue; often there is only constipation with the passage of mucus and hardened feces. In an obscure case a study of the stools should be made, as the detection of amœbæ in the mucus or in the feces may point to the true conclusion.

Locally there is pain over the liver, which may be referred to the region of the right or the left lobe. It may be seated in the fifth or sixth interspaces anteriorly, or at the ninth and tenth ribs posteriorly. There may be pain in the right shoulder mechanism. The pain may be paroxysmal, or it may be intense and persistent.

Physical Signs. (Plate XLII., Fig. 1.) The enlargement may be uniform if the abscess is central; on the other hand, the enlargement may extend chiefly upward in the anterior, the axillary, or the posterior region. When the convex surface of the right lobe of the liver is affected, the enlargement is usually upward. An abscess in the lower portion of the right lobe produces a downward enlargement, and the lobe of the liver

FIG. 379.



Intermittent fever in abscess of the liver.

can readily be detected on palpation. When the abscess is limited to the left lobe of the liver, and is situated about the suspensory ligament, the enlargement may be seen below the xiphoid cartilage. The mass may extend to the umbilicus and project forward; sometimes it is sufficiently large to cause eversion of the ribs of each side and render the entire epigastrium unusually prominent. The surface may become reddened and œdematous. Over the tumor, which is hard at first but later becomes soft, there is tenderness on palpation, and there may be, as in other situations, fluctuation.

The irregular enlargement is made out by percussion. The outline may be difficult to map out on account of secondary pleural effusion, or secondary pleural inflammation with the development of hepatopulmonary fistula, causing dulness posteriorly. If the case has been seen from the first, a friction-sound may be heard, followed by the physical signs of effusion. The *x*-rays have been successfully used to determine the presence of an irregular enlargement of the liver.

Atypical cases are characterized by the absence of general symptoms or local signs. Fever may be absent entirely, exhaustion alone being present, which might be accounted for by the previous dysentery. Pronounced anæmia due to the dysentery may be the most marked symptom; inflammation of the joints, or neuritis, may be present. In a case under my care the only symptom for a long time, with the exception of anæmia and loss of appetite, was severe pain in the sixth interspace. In other instances there are no liver-symptoms whatsoever. General symptoms of infection, or an irregular, or even a continued fever, the cause of which can not be ascertained, may alone be present. In one of my cases there was moderate continued fever, with loss of appetite and dyspeptic symptoms; there was no diarrhœa; no cause could be found for the fever, although a slight enlargement of the liver was noted. The patient slipped out of the ward and went down into the yard to smoke; on his return he was seized with an intestinal hemorrhage which could not be checked and resulted fatally. At the autopsy a large abscess of the liver was found and there was ulceration of the rectum, from which the intestinal hemorrhage had taken place.

Perforation of Hepatic Abscess. The first intimation of the existence of an abscess is sometimes obtained from the occurrence of rupture. If perforation takes place into the peritoneum, it is not likely that the cause can be determined during life. The abscess frequently ruptures through the diaphragm into the pleura, and thence into the lung. An empyema may be set up, the true source of which may not be ascertained unless the pus is examined. The physical signs are those of empyema—dulness or diminished resonance, absence of fremitus and vocal resonance, diminished breath-sounds, impaired movement, cough, and dyspnœa. When the lung is infected, the physical signs may resemble those of consolidation. We find dulness, bronchial breathing, and increased tactile fremitus. A harassing, convulsive cough occurs, and sooner or later expectoration of a reddish-brown, brickdust-colored material resembling anchovy sauce. This characteristic expectoration is decisive. It contains amœbæ, and, in addition to blood-pigment and corpuscles, orange-red crystals of hæmatoidin, cholesterin-plates, and leucin and tyrosin. When the abscess perforates into the stomach or bowel, the discharge from either may be of the above-mentioned nature. Perforation into the pericardium is usually followed by immediate death.

Diagnosis. The diagnosis is usually not difficult in typical cases. Under all circumstances attention must be paid to the facts bearing upon the ætiology, and to the association of general and local symptoms. In the presence of general symptoms of suppuration hepatic abscess may be mistaken for *malaria*. Examination of the blood and the result of treat-

ment by quinine would establish a diagnosis of malarial fever. It is difficult sometimes to determine whether the abscess is in the abdominal wall or in the liver proper, or whether it is situated beneath the diaphragm. If the liver is movable with respiration, the two former conditions may be excluded. A *mural abscess* is not influenced by respiration, while in subdiaphragmatic abscess the movement is impaired. *Suppuration of a hydatid cyst* can not be distinguished unless it has been known beforehand that a simple hydatid was present in the liver. Under such circumstances, if suppuration occurs, it is likely to be confined to the cyst. Abscess of the liver must be distinguished from *gallstones* attended by intermittent fever without suppuration. While the distinction is difficult in many cases, yet the history of the case, the association of jaundice becoming intensified after each paroxysm, and the good general nutrition of the patient point to gallstones. Abscess of the liver is of shorter duration than cholelithiasis, and its primary cause can usually be ascertained by examination of the rectum, or the discovery of suppuration in other parts of the body.

Aspiration. Exploratory puncture must be employed in many cases, and it can usually be done with safety. The region in which the enlargement is greatest, or at which the swelling is most prominent is the point of election. In abscess secondary to dysentery a brownish-colored pus resembling anchovy sauce will be withdrawn. It may be of a peculiar odor, and, on examination, amœbæ may be found. If there is no point of election, the needle may be introduced in the lowest interspace in the anterior axillary, or the seventh interspace in the midaxillary line. A fairly large-sized needle should be used. Pus may be present, and yet not be reached by aspiration.

Suppurative Pylephlebitis.

Abscess of the liver may be due to *pyæmia*. It may be a part of general or of portal pyæmia. Parasites and foreign bodies, as well as gallstones, may excite an abscess. The echinococcus cyst may suppurate, or round worms may penetrate to the liver and cause suppuration.

The symptoms of *suppurative pylephlebitis* and of *pyæmic abscess* are general and local. Jaundice is more common than in solitary abscess; pain and tenderness are more pronounced over the liver, which is uniformly enlarged. With the enlargement of the liver and jaundice we have the usual symptoms of pyæmia. Sometimes the fever is distinctly intermittent; at other times it is irregular and septic in character.

Cirrhosis of the Liver.

A diffuse interstitial inflammation of the liver, frequently with atrophy of the organ, which in the large majority of cases is caused by irritants entering the portal circulation through the stomach. Among these irritants alcohol is the most common, particularly the stronger liquors, as gin and whiskey. Other irritants, as spices used to excess, may likewise cause the diffuse inflammation. Cirrhosis of the liver may also

be a sequel to the infectious diseases, notably scarlatina, and may be excited by malaria. The most frequent result of alcoholism upon the liver is the *atrophic* form of cirrhosis. *Hypertrophic* cirrhosis is a common disease, however, and is usually due to alcoholism, though it bears a closer relation to infections than does the atrophic form. There is good evidence that toxic matters of various kinds other than alcohol, when absorbed from the digestive tract, lead to cirrhosis of the liver, and it is uncertain whether the substances contained in the alcohol itself or toxic substances produced by the gastro-intestinal disturbance which the alcohol sets up cause the interstitial over-growth.

Hypertrophic biliary cirrhosis is due to obstruction of the bile-ducts, followed secondarily by over-growth of the connective tissue. Cirrhosis of the liver may arise in the course of *syphilis*; and finally a secondary cirrhosis of the liver which develops in the course of passive congestion of that organ is described under the name of *nutmeg-liver*.

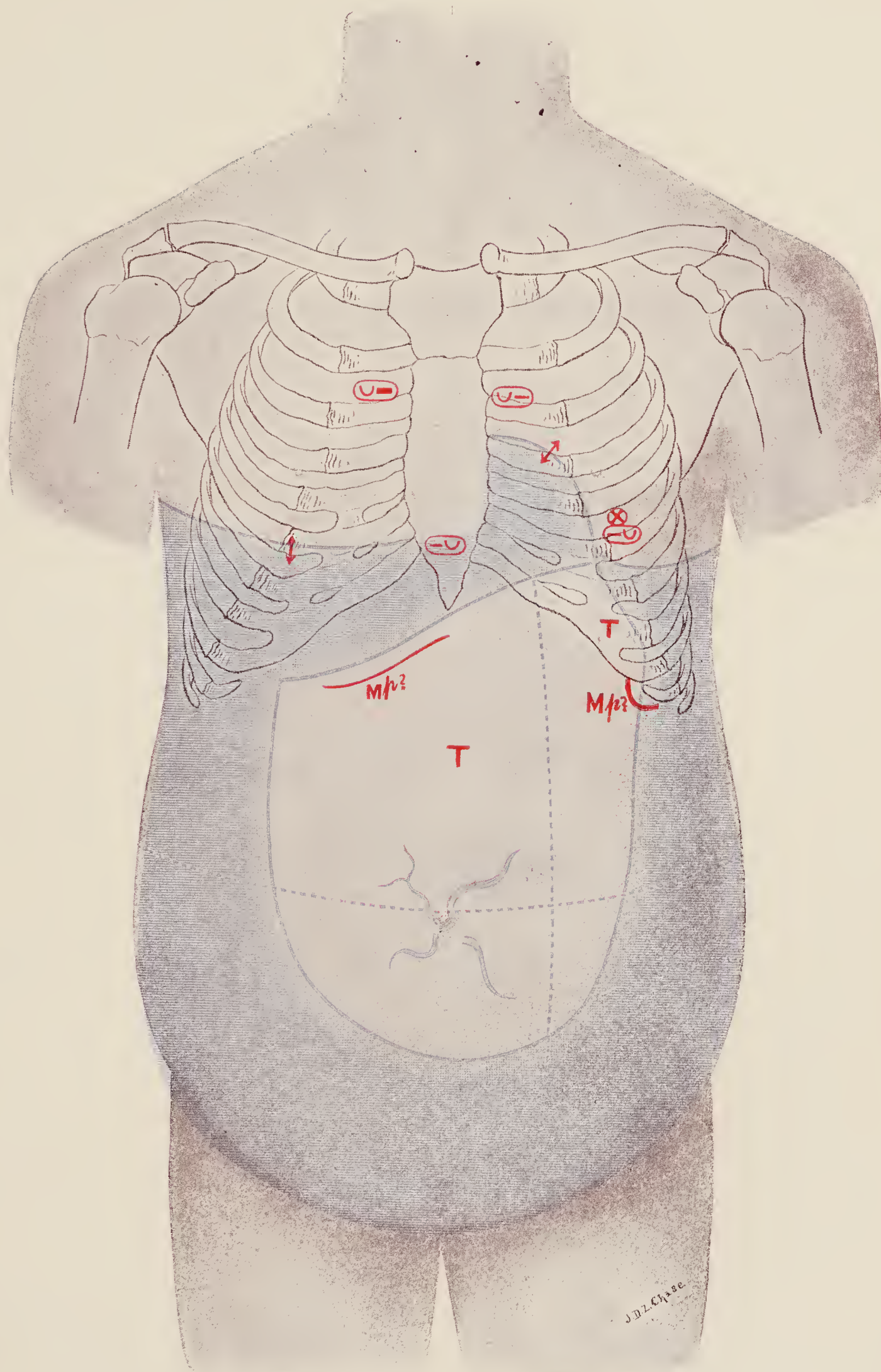
Atrophic Cirrhosis. Cirrhosis of the liver of the atrophic form, due to alcohol, presents various clinical features. In the first place, it may exist without causing any symptoms whatever during life, and be discovered after death from other causes; or it may present no symptoms until an accident occurs in the course of the disease, as hemorrhage from some portion of the collateral circulation. In both cases the symptoms are absent because the collateral circulation is complete. If this is incomplete, however, grave local and general symptoms ensue. Before detailing these symptoms it may be well to state that the occurrence of one symptom, which we have termed accidental, may lead to the inference that cirrhosis of the liver is present, particularly if the patient has been an alcoholic. This symptom is *hemorrhage*. It may be from the stomach, causing death at once or after repeated loss of blood; it may also take place from the intestine.

Symptoms. The symptoms are *general*, due to interference with the nutrition; and *local*, their extent depending upon the degree of obstruction to the portal circulation. They have been divided into those of the *first stage*, or stage of enlargement, and those of the *second stage*, or stage of contraction. This division is, however, not usually recognized at present. There is no real stage of hypertrophy; and while congestive enlargement does sometimes occur, it is by no means constant. Cheadle's experience is most convincing on this point. There are, however, two sets of symptoms.

During the *first stage* the symptoms are those of gastritis with enlargement of the liver. The gastric symptoms are morning retching or vomiting, with discharge of mucus, associated with other symptoms of gastric catarrh, as loss of appetite, nausea, tenderness in the epigastrium, eructations, constipation, and loss of flesh and strength. The liver may be found a little swollen.

The symptoms of the *second stage* are more severe and are due to obstruction of the portal capillaries. On examination *ascites* is detected. The transudate may be enormous, causing monstrous distention with pouting of the umbilicus. The *spleen* is found to be enlarged, extending over twice or three times the normal area of percussion. If ascites does

PLATE XLIII.



Cirrhosis of the Liver with Ascites.

not interfere, the edge of the spleen can be readily made out. The portal obstruction causes secondary gastro-intestinal catarrh, if it was not already present on account of the alcoholism. Although constipation is usually present, there may be persistent diarrhœa, usually lenteric and occurring in the morning only. Hemorrhages may take place at any time from the gastro-intestinal tract, either from the stomach or from the intestine. Not infrequently they occur from the œsophagus on account of varicosity of the veins at the junction of the œsophagus and cardiac end of the stomach. Hemorrhoids are always present and may bleed at each stool. Jaundice is not the rule, and, if present, is usually slight and due to the duodenal catarrh.

The **general symptoms** of cirrhosis, and particularly the symptoms of the later stages, are striking and diagnostic. The nutrition is much impaired. The patient who, in the large majority of cases, had been corpulent, becomes emaciated. The skin changes in color and becomes of an earthy-gray or dirty sallow hue. The capillary venules of the face are dilated; the distended capillaries on the nose are distinct. Later, ecchymoses may occur in the skin, and hemorrhages take place from the mucous membranes and into the retina. Debility ensues; œdema of the ankles is almost sure to occur, and sometimes general anasarca may take place. It is extremely rare to have fever unless complications occur. The pulse is small and becomes more rapid than normal; the heart-sounds grow weaker. The skin may be the seat of eruptions, and chronic skin diseases of various kinds develop.

The *urine* throughout the disease presents nothing characteristic; as ascites develops, it becomes scanty and dark, and loaded with urates and uric acid. In rare instances it may contain sugar, and if the uric acid is in excess, albumin.

The *collateral circulation*, which develops in order that the portal blood may reach the right heart, takes place in various ways. First, communication may be formed between the veins of the mesentery and those of the posterior abdominal walls; second, between the coronary veins of the stomach and the veins of Glisson's capsule and the phrenic veins; third, between the hemorrhoidal and the inferior mesenteric veins; fourth, between enlarged veins occupying the position of the obliterated umbilical vein in the ligamentum teres, and the epigastric and mammary veins.

Physical Signs. (Plate XLII., Fig. 2, and Plate XLIII.) It may be difficult to elicit the physical signs before paracentesis is performed on account of the extensive ascites. The liver will be found to have undergone contraction; although diminution in the area of dulness is not by any means so absolutely confirmative of contraction as the opposite condition is of hypertrophy. Percussion should be performed several times, because the distended intestinal coils may obscure the findings. A well-defined caput Medusæ is often present.

Diagnosis. The diagnosis is usually not difficult if the complete picture of the case is presented. It can not be established positively without definite knowledge of the cause. If the patient comes under observation after ascites has developed, the diagnosis is more difficult. It must, in the majority of cases, be based upon exclusion of heart, lung, and kidney

disease. A history of alcoholism and the presence of other symptoms of liver disease point to the hepatic origin of ascites. Ascites may be due to other causes within the abdomen, notably *chronic peritonitis*, exclusion of which is sometimes difficult. General tenderness, absence of distention, and splenic enlargement point to peritonitis. The *fatty cirrhotic liver* may present symptoms similar to those of the atrophic form, except that the organ is enlarged.

In a study of a case of cirrhosis of the liver a judgment as to its nature may be, in a measure, confirmed by the presence of other phenomena due to the same cause. Very frequently we have, at the same time, cirrhosis of the kidneys and sclerosis of the arteries, with secondary atheroma, both of which have led to hypertrophy of the heart. Strümpell refers to the association of cirrhosis and chronic tuberculous peritonitis, and thinks the former is the primary lesion.

The *duration* can not be determined accurately, as the onset is usually insidious. After the ascites appears the duration may vary from six to eighteen months, depending largely upon the completeness of the compensatory circulation. Death usually occurs from intercurrent disease or progressive exhaustion. In not a few cases cerebral symptoms occur. In addition to the cirrhotic cachexia, the sudden occurrence of coma and convulsions, preceded by delirium, may ensue; the cause of this is not fully known. It must be borne in mind that the occurrence of these symptoms in an alcoholic subject may be due to a cirrhosis, the presence of which had not been suspected during life.

Hypertrophic cirrhosis, or so-called biliary cirrhosis, presents a somewhat different picture. In the first place, there often is a history of gallstones, or obstruction of the duct from other causes. The patients are frequently young adults, and in general the age of the subjects is less than in the atrophic form. The liver is uniformly enlarged, and the surface is smooth and strikingly indurated. There are weakness and loss of appetite. Jaundice ensues very early, or may be the first symptom. It increases and persists throughout the course of the disease. Ascites is somewhat less common than in the atrophic form and is often very slight or absent altogether; only very rarely, indeed, does it become a pronounced symptom. The enlargement and jaundice may continue for months or even years without the development of grave symptoms.

Fever may, however, set in at any time, being in all probability due to the biliary obstruction. It comes on in distinct paroxysms associated with increase of jaundice. Sometimes distinct increase in the enlargement of the liver and exacerbation of the general symptoms grow temporarily quite marked; the temperature rises to from 102° to 104° F. In some cases the tongue becomes dry and brown, the pulse rapid, and all the symptoms of grave febrile jaundice ensue, the case ending fatally, with severe nervous symptoms. At any time during the course of the disease the patient may be seized with convulsions, followed by coma and death. It is now generally considered that in this form of cirrhosis the liver remains large; in other words, that there is no tendency to ultimate contraction. There is, however, not uncommonly more or less ascites, though the amount of fluid is often so slight as to escape clinical obser-

vation. Enlargement of the spleen is very common, and is probably due rather to a participation of the spleen in the cirrhotic process than to passive congestion from portal obstruction.

The **diagnosis** is often difficult. Gradual and persistent jaundice continuing for a long time without cause, and associated with persistent enlargement of the liver without symptoms of portal obstruction in a non-alcoholic subject, points pretty certainly to hypertrophic cirrhosis of the liver.

Syphilitic Disease of the Liver.

Syphilitic disease of the liver may result either in cirrhosis or in the development of gummata. *Syphilitic cirrhosis* presents the same symptoms as the alcoholic form. The history, the marked irregularity on the surface of the liver, and the existence of syphilis elsewhere may lead to a diagnosis of the true condition.

In *congenital syphilitic disease* of the liver the inflammation is diffuse; the liver is enlarged and hard; the surface is smooth; there are usually syphilitic lesions in other organs; the patient presents syphilitic eruptions, and has the well-known wizened appearance that belongs to the disease.

Syphilitic gummata in the liver may exist without presenting any symptoms whatsoever, or they may reveal their presence by pain and a localized swelling and discomfort which call the patient's attention to the region, particularly if his general health is reduced at the same time. Tumors are situated in the left lobe, in the median line, or along the margin of the ribs. The pain is usually localized in this region, but may extend more or less over the entire liver, particularly if there is general perihepatitis along with other evidences of syphilis; the latter are not always present, however. If the temperature is taken frequently, a moderate febrile range will be observed. It may not rise above 100.5 ° F.; but in the absence of other causes this is a valuable diagnostic symptom.¹ In other instances the gummata may grow in such a situation as to interfere with the portal circulation or press upon the gall-ducts. The latter is very rare. Gummata that are accessible to palpation appear as enlarged bosses, giving the sensation of flattened hemispheres. Sometimes several separate elevations can be made out on the surface of the enlarged organ. To determine the exact nature of the lesion is often very difficult. The symptoms may point conclusively to hepatic disease. Knowledge of the presence of syphilis aids in the diagnosis. If without a syphilitic history there are scars in the throat, nodes on the bones, or other signs of syphilis, the diagnosis will be tolerably certain. Severe pain is more prominent in syphilis than in cirrhosis, and the nodules of syphilis are very different from the granular surface of cirrhosis. Amyloid disease is often associated with the actual syphilitic lesions, and much of the enlargement may be due to this and persist after treatment has caused absorption of gummata.

¹ "The Diagnostic Importance of Fever in Late Syphilis." Musser, University Medical Magazine, October, 1892.

Fatty Liver.

The symptoms of fatty liver are not marked. The physical sign is a uniform enlargement extending in all directions. On palpation the edges can be felt; they are rounded and smooth. They are soft at first, but later become indurated. Fatty liver may be followed by cirrhosis after a period of alcoholism. The general symptoms are those of the primary disease. Fatty liver occurs in gouty subjects, but is notably present in wasting diseases, in tuberculosis, in chronic hip-joint disease, and in amyloid disease of the liver.

Fatty liver sometimes follows the congestion of the liver which is present in the course of organic heart disease. It is not a true fatty liver, but a fatty cirrhosis. There is increased fatty degeneration with an over-growth of connective tissue. This form is associated with heart and kidney disease. On palpation the edges of the liver are indurated. The liver may undergo diminution in size later, and the symptoms of cirrhosis ensue.

Amyloid Disease of the Liver.

Enlargement of the liver without pain is often due to amyloid disease. Similar disease is found in other organs, and there is present, to point to the nature of the enlargement, syphilis, bone disease, prolonged suppuration, or tuberculosis. In amyloid disease the pallor of the patient is great; the face may be swollen, and the ankles slightly œdematous. The spleen is enlarged, the urine albuminous and abundant, but of moderate specific gravity. A history of syphilis is an important point in establishing the diagnosis. Fatty liver can readily be distinguished from amyloid disease by palpation. In the latter the surface is smooth, but very much indurated.

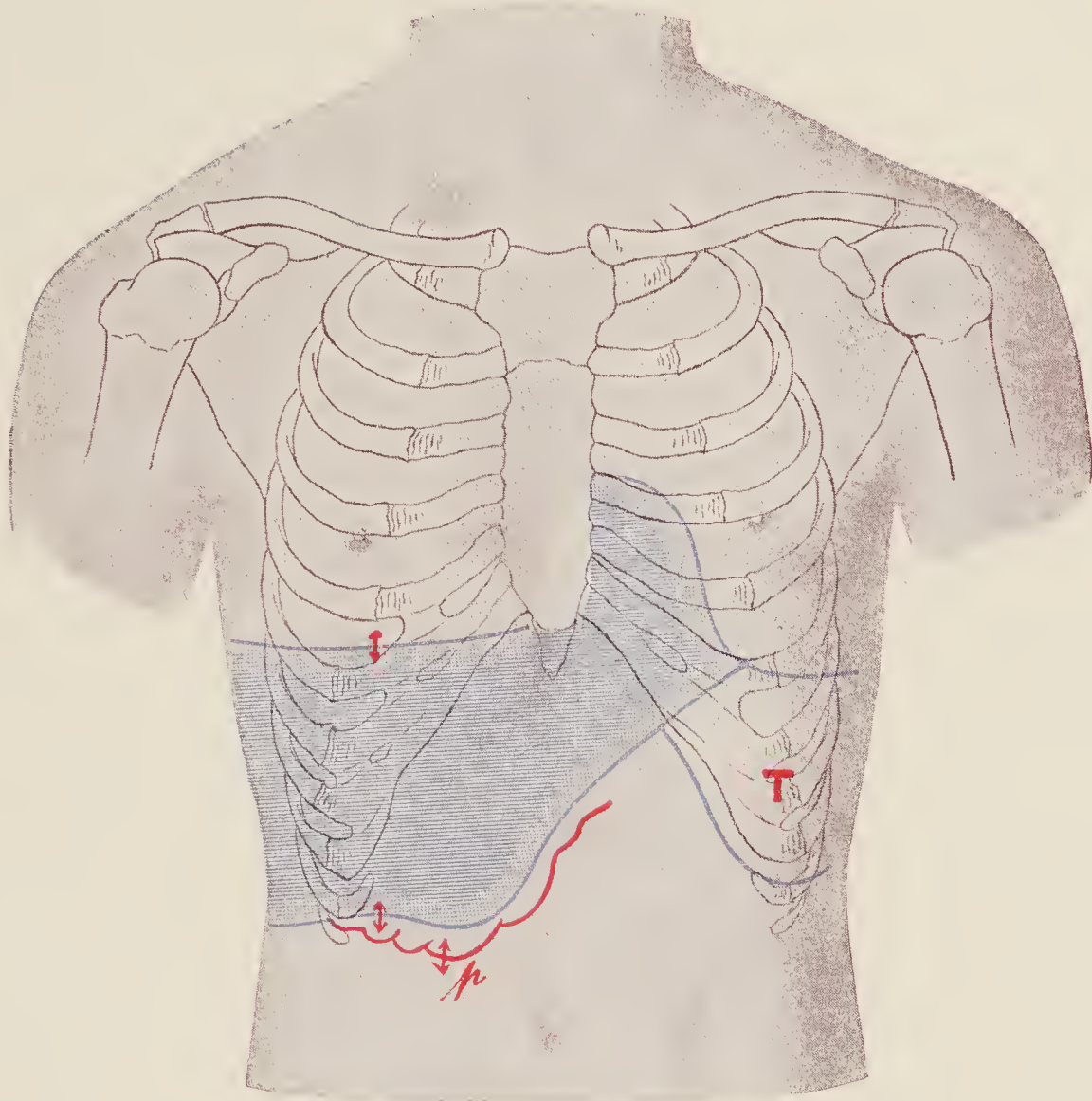
Cancer of the Liver.

The ætiological factors upon which the diagnosis of cancer is based are: the age of the patient—most frequently between the fortieth and sixtieth year; the sex, women being more frequently attacked; and heredity. The disease is nearly always secondary to cancer in some other situation; consequently, in cases in which symptoms point to cancer of the liver, search must be made for the primary lesion elsewhere. The most frequent seats are the rectum, the uterus, the stomach, and the remainder of the gastro-intestinal tract. Sarcoma of the liver, chiefly of the melanotic variety, is not uncommonly secondary to melanotic sarcoma of the eye. Further ætiological influences that may bear upon the diagnosis are: (1) the occurrence of gallstones, which act as the exciting cause in the development of primary cancer of the ducts, thence spreading to the liver; (2) the occurrence of trauma.

Symptoms. The symptoms of cancer of the liver may be due to increase in the size of the liver; to pressure of the growths upon the ducts or terminal portal vessels; and to the general effects of carcinoma upon the system—the *cachexia*. They are, *jaundice*, which is not very deep unless the common duct is affected; *ascites*, which is always present

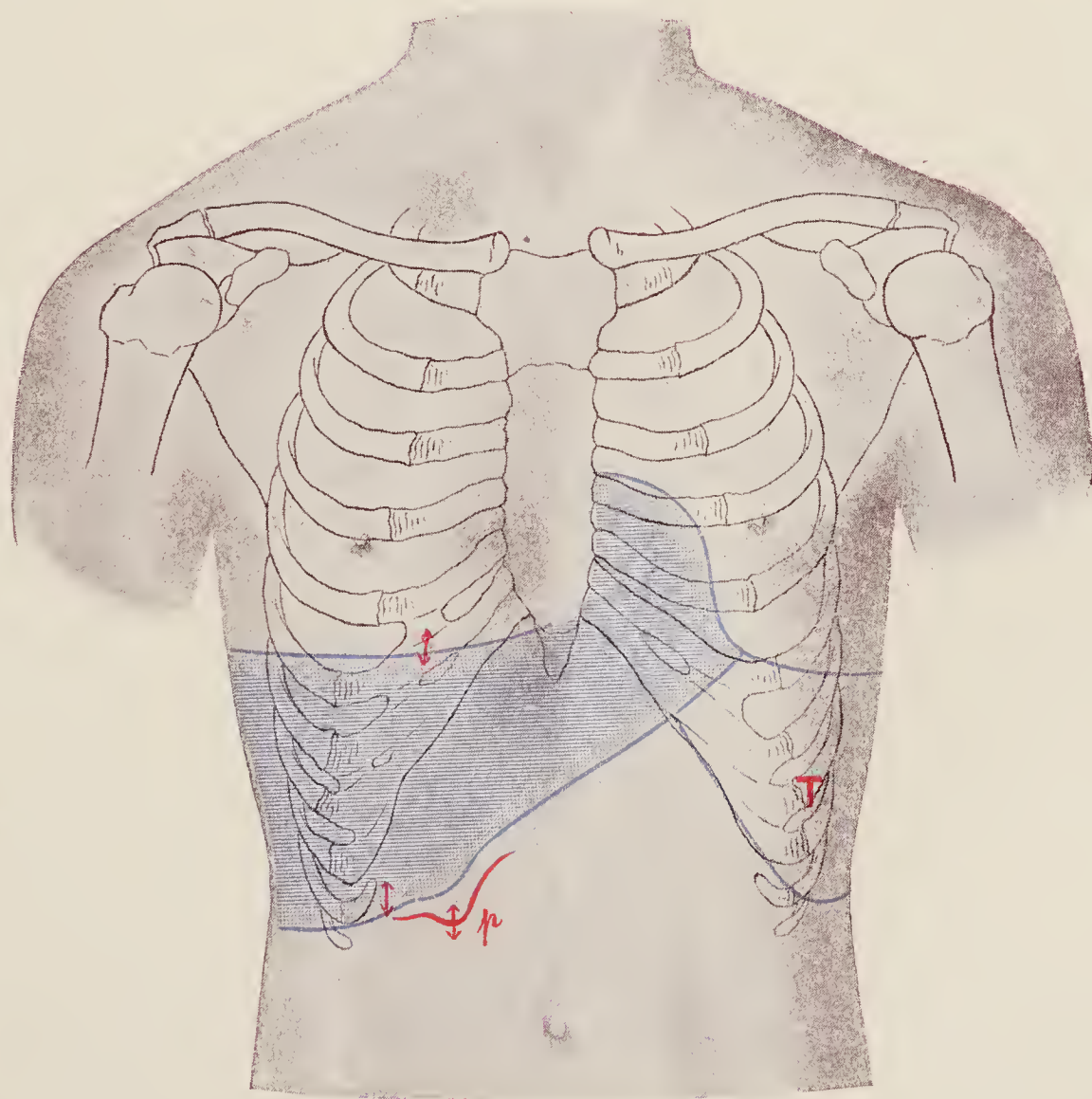
PLATE XLIV.

FIG. 1.



Carcinoma of the Gall Bladder with Involvement of the Liver.

FIG. 2.



Enlargement of the Gall Bladder.

in the atrophic forms, but may be absent when the liver is enlarged; the general symptoms of rapid *emaciation* and prostration; and in some instances *moderate fever*. Fever attends the rapidly growing cases; it is usually continuous, but may be intermittent, especially if there is supuration or suppurative inflammation of the ducts. It is a well-known fact that *gallstones* are of common occurrence in patients suffering from cancer in any location whatever. The symptoms of biliary calculus or of obstruction may attend those of secondary cancer of the liver, and the stone has an ætiological significance.

In many instances *secondary cancer* of the liver may be present without symptoms to attract attention to this organ during life. If cancer in certain other regions has continued for the usual period of time, it is almost certain that at the autopsy cancer of the liver will be found.

Physical Signs. (Plate XL., Fig. 2; Plate XLII., Fig. 2; and Plate XLIV., Fig. 1.) The liver is enlarged and its surface irregular. By palpation the organ can be made out extending below the margin of the ribs. The edges are irregular, and bosses can be distinctly felt on the surface. In rare cases one or two masses only may be present, growing out of the substance of the left lobe of the liver and causing a large tumor below the sternum. The nodules are usually hard, but sometimes may be soft and even fluctuate. After emaciation becomes marked, the nodules can be seen as well as felt near the surface of the skin, and their number distinctly made out. The abdomen is distended.

The liver moves with respiration. Progressive enlargement can be noted while under observation. The enlargement can be well defined by percussion; and while the surface is irregular, the general shape of the dulness corresponds to that of the liver. The increased size and inflammation of the capsule cause a sensation of weight in the hepatic region and pain which may be intermitting in character. The nodules may be tender on palpation. The superficial veins are enlarged.

In not every instance do we find enlargement. In some cases the cancer is associated with cirrhosis of the liver, or may itself be of nodular type, and in the course of the disease undergo shrinkage. The liver is then normal or diminished in size.

Diagnosis. The diagnosis of cancer of the liver is not difficult when the changes in the liver can be made out on palpation and percussion. In rare instances, in which the liver is smooth, it may be mistaken for fatty or amyloid liver. A definite cause can usually be assigned for the latter; while the occurrence of jaundice, the rapid increase in the size of the liver, and the general symptoms of the cancerous cachexia indicate cancer of the liver. A *syphilitic liver* with irregular gummata may cause serious doubt; the history of the case and other signs of syphilis aid in the diagnosis, although locally the condition may exactly simulate carcinoma. The jaundice, however, is neither so frequent nor so deep in syphilitic gummata, and the cachexia does not ensue; but the therapeutic test may be necessary to make a diagnosis.

In *hypertrophic cirrhosis* of the liver the jaundice is deep and the liver is enlarged; but there is little wasting or anæmia. The surface of the liver is smooth; there are certainly no bosses, and the organ is painless.

Ascites is more common in cirrhosis; the patient is usually affected earlier in life than in cancer.

In a large growing cancer one or two of the nodules may suppurate and simulate *abscess of the liver*. Abscess follows a definite cause usually, and occurs in middle life; cancer is secondary to disease in other organs and occurs usually in late life. The results of aspiration differ in each. Moreover, a history of dysentery, the occurrence of pain, of profound anæmia, of pronounced hectic fever with irregular enlargement of the liver, but without jaundice or cachexia, point to abscess.

Cancer of the liver may be simulated by cancer of organs in close proximity to the liver, as the *pancreas*, the *pyloric end of the stomach*, or the *colon*. In addition to the fact that the usual symptoms of pyloric cancer are present, it will be found that jaundice occurs late. Cancer of the pyloric end is less freely movable with respiration unless it becomes adherent to the liver. *Cancer of the pancreas* also presents difficulties; a tumor in the midcostal region, however, with vomiting and the early development of jaundice, *before* the liver has become enlarged or nodular, and associated with other characteristic symptoms, such as intestinal dyspepsia and fatty stools, points to the pancreas as the primary seat of the disease. *Cancer of the omentum and colon* are not modified by respiration. The percussion-note over them is different; they frequently extend beyond the liver-confines and are associated with symptoms of obstruction of the bowels. *Fecal accumulation* in the transverse colon must not be mistaken for cancer of the liver. The large masses adjacent to the liver may closely simulate cancerous nodules. In doubtful cases the colon should be emptied.

Cancer of the liver and hydatid disease must not be confounded. The tumor in *hydatid disease* is usually single; it is large, and may fluctuate or yield the hydatid fremitus. It causes irregular enlargement of the liver when the tumor presents in the epigastrium or along the margin of the ribs. It is painless. Aspiration yields the characteristic hydatid fluid.

Cancer of the bile-ducts can not always be distinguished from cancer of the liver. Jaundice early in the course of the disease, in a person who has had gallstones, followed by enlargement of the liver and gall-bladder, in the absence of primary disease elsewhere, suggests cancer of the gall-bladder or ducts. This is more or less confirmed if the smooth and painless gall-bladder becomes hard, irregular, and tender on pressure.

Hydatid Disease of the Liver.

Hydatid disease is comparatively rare in this country, but, in my experience at least, it is undoubtedly increasing in frequency. Without any increase in the opportunities for observation, I have seen seven cases within the last two years, compared with the same number during the five preceding years. The disease occurs in people who live with dogs. It may occur at any age, but is most common in adult life. It is very rare before the fifth year.

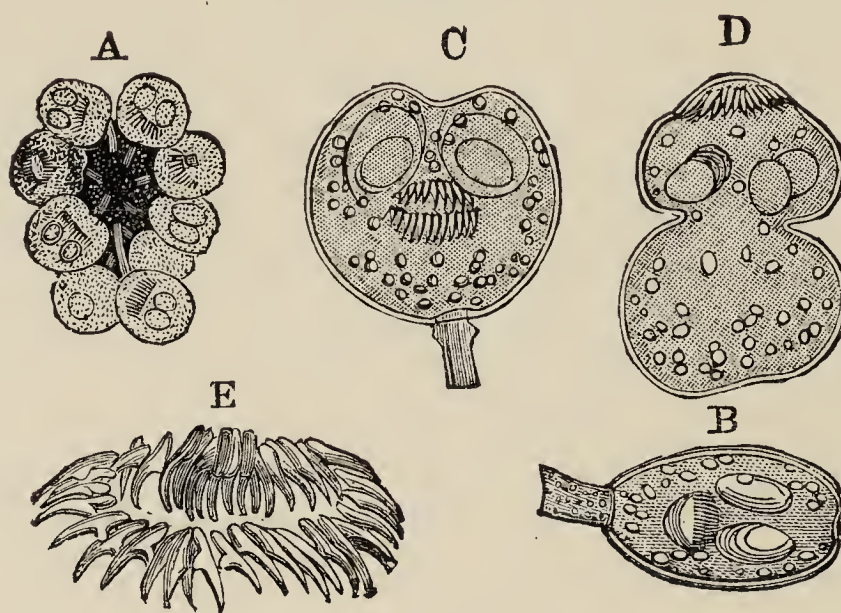
Symptoms. The *general symptoms* are negative; the nutrition does

not suffer unless the enlarged mass interferes, by its pressure, with the physiological acts of digestion and assimilation. If suppuration sets in, the general symptoms of abscess of the liver arise. Jaundice is more common than in tropical abscess. The abscess may perforate into one of the adjacent hollow viscera, or into the pleura and bronchi; it may perforate externally; and finally it may perforate into the pericardium or vena cava, and cause death. If perforation takes place in the biliary passages, obstructive jaundice arises, with secondary suppurative cholangitis. Urticaria sometimes follows rupture or aspiration of the cysts. This is not of diagnostic significance, except that it may point to rupture of the cyst. The *local symptoms* depend upon the size of the *tumor*. Small cysts may be present without causing any disturbance. Large and growing cysts cause signs of tumor, with great increase in the size of the liver. The *physical signs* depend upon the situation of the tumor. It may be found in the median line above the umbilicus, causing a distinct prominence, tense and firm, which sometimes yields fluctuation. Quite often the tumor grows at the suspensory ligament, pushing the diaphragm upward, dislocating the heart and causing an increased area of dulness in the left upper quadrant. In this position it may simulate a pancreatic cyst or effusion into the lesser peritoneal cavity. If the tumor is in the right lobe, the enlargement of the liver may be upward or downward. The upper border of liver-dulness may begin two or three interspaces higher than normal posteriorly or in the axillary region. If the cysts are superficial, when palpated with the fingers of the left hand and percussed with the right, a vibration or trembling movement is felt, which may continue for a certain time. It is known as the *hydatid fremitus*. It is not always present. The enlargement is painless. Local sensations of weight and dragging may be complained of. If suppuration sets in, there may be a good deal of pain.

Diagnosis. The diagnosis is not difficult. The occurrence of irregular painless enlargement of the liver without general symptoms is significant. If fluctuation or the characteristic fremitus is detected, a more positive conclusion can be reached. When suppuration takes place, the symptoms are like those of abscess of the liver. Hydatid disease is to be distinguished from *syphilitic hepatitis*, in which the enlargement is hard and irregular, and does not fluctuate. Sometimes the symptoms resemble cancer; but the age of the patient, the presence of jaundice, and the extreme emaciation and cachexia indicate that affection rather than hydatid disease. *Enlargement of the gall-bladder* containing a mucoid fluid, in which fluctuation can be detected, may simulate hydatid disease. The enlargement, however, may be preceded by conditions causing obstruction of the cystic duct. The gall-bladder is movable. In some instances there may be resonance between it and the liver. It is usually of a pyriform or oblong shape. In *hydronephrosis* the symptoms of a localized cyst are present, which does not, however, move with respiration, as in hydatid disease. Exploratory puncture is sometimes necessary to establish a diagnosis. A hydatid cyst is frequently confounded with *pleural effusion* of the right side, for there may be all the physical signs of effusion at the right base. The distinction can be made by the char-

acter of the line of dulness. In hydatid cysts, as Frerichs points out, the line corresponds to a curve having its greatest height in the scapular region. It is not difficult usually to distinguish hydatid cyst from other

FIG. 380.



Human echinococci. (From FINLAYSON, after DAVAINÉ.)

- A, a group of echinococci, still adhering to the germinal membrane by their pedicles. $\times 40$.
 B, an echinococcus with head invaginated in the body. $\times 107$.
 C, the same compressed, showing the suckers and hooks of the retracted head.
 D, echinococcus with head protruded.
 E, crown of hooks, showing the two circles. $\times 350$.

forms of painless enlargement. In *fatty* and *amyloid* disease the enlargement is uniform. Both occur more commonly in individuals who have been in ill health, whereas hydatid disease occurs in healthy individuals.

FIG. 381.

Hooks from *Tania echinococcus*. $\times 350$. (CORTI.)

An absolute diagnosis of hydatid disease is based upon the results of *exploratory puncture*. When this is made over a tumor, or the centre of dulness, if it is due to hydatid disease, a clear, slightly opalescent fluid is withdrawn. The fluid has a specific gravity of 1005 to 1009, is of neutral reaction, and does not contain albumin but does contain chlorides and sometimes traces of sugar. Hooklets may be found in the clear fluid.

Displacements of the Liver.

The literature contains about 80 cases, in many of which disease of the gall-bladder or gall-ducts was simulated because of (1) attacks resembling gallstone colic caused by obstruction of the ducts, (2) obstructive jaundice from kinking of the bile-ducts, and (3) the presence of a tumor simulating an enlarged gall-bladder.

Diagnosis. The diagnosis is based on (1) the association with other displacements, and the patient's physique—long-chested subjects are predisposed to displacement of organs; (2) the clinical course; and (3) the physical signs.

Clinical Course. Most of the cases occur in women, and there is usually a history of trauma or abdominal disease. The patients usually suffer from neurasthenia and gastro-enteroptosis. The condition is one of long duration marked by recurrent attacks of pain, transient jaundice, bilious vomiting, and symptoms due to pressure upon other organs; fever is absent except in a few cases.

Physical Examination. Diastasis of the recti muscles is frequently present. On palpation a tumor of the size, shape, and consistence of the liver is discovered in the abdomen; it moves with respiration like the liver, and can be replaced by palpation, or returns to the normal position when the patient assumes the recumbent posture. The signs are readily brought out with Glénard's *procédé du pouce* (*q. v.*). On percussion, which must be performed in the erect as well as in the recumbent posture, a tympanitic note is obtained over the normal area of liver-dulness, except in anteversion when the convex surface is in contact with the diaphragm and anterior abdominal wall, and the note is therefore dull.

Deformities of the Liver.

Floating Lobes, Riedel's Lobe. The abnormally elongated lobe forms a tongue-like process which sometimes simulates tumor of the gall-bladder.

Clinical Course. The clinical course is uneventful, or there may be occasional attacks of pain and tenderness coincident with the presence of the tumor, which disappears at times. Women are chiefly affected, especially such as have been addicted to the practice of abdominal constriction. Attacks of gallstone colic and cholangitis may accompany, or in rare cases follow the condition.

Physical Examination. The tongue-like enlargement is smooth and solid to the touch, of variable size, and movable by palpation and with respiration; in rare instances it behaves like a floating kidney. The tumor disappears at times, and it may or may not be possible to demonstrate by percussion its connection with the liver. The percussion-note is dull when the tumor is thick; but it may be so thin that the resonance of the underlying hollow structures is not obscured.

Diagnosis. *Cholecystitis* may be simulated, as the tumor is at times the seat of pain and tenderness. *Cholelithiasis* is a frequent accompaniment, and the coincidence may give rise to confusion.

Corset Liver. This deformity may also simulate tumor of the gall-bladder.

Clinical Course. The clinical course resembles that of floating lobe and is marked by hepatic incidents. Corset liver may be associated with cholecystitis, cholangitis, cholelithiasis, gastro-enteroptosis, and carcinoma.

Physical Examination. The tumor is not unlike that caused by a floating lobe. When separated from the liver it is variable in size and shape, of the consistence of the liver, and movable to an extreme degree. The abdomen is tender and swollen. The tumor is always superficial, is never found in the loin, and is sometimes the seat of spontaneous pain.

DISEASES OF THE GALL-BLADDER AND GALL-DUCTS.

Inflammation of the Gall-ducts. Catarrhal Cholangitis.

Causes: secondary to gastroduodenal catarrh, to pressure, to local spreading infections. Other cases of catarrhal jaundice are found to be primarily infectious.

Symptoms. The symptoms and signs are well known. The age of the patient, the presence of a cause, and the clinical course make up the picture. When long continued, catarrhal inflammation may resemble obstruction due to other primary or secondary processes.

Diagnosis. The diagnosis in cases of so-called catarrhal jaundice continuing more than six weeks should be revised if the number of erythrocytes suddenly diminishes, the hæmoglobin falls, the spleen enlarges, and there is loss of weight. The true nature can be determined by the antecedents or by accompaniments, which by this time may be more prominent, as the enlarged glands of syphilis or tuberculosis. Organic diseases, cirrhosis, cancer of the liver, and infections, as Weil's disease, must be excluded. Many so-called catarrhs are the result of typhoid, pneumococcus, or other infections.

Suppurative Cholangitis.

The conditions for which this disease may be mistaken are pyelophlebitis, abscess and syphilis of the liver, rare cases of cancer, and Weil's disease.

The diagnosis is based on the:

1. **Clinical Course.** Gradual onset; infection of ducts or gall-bladder; presence of foreign bodies (gallstones); history of a previous general infection.

2. **Objective Diagnosis.** Icterus may or may not be present. The fever is characteristic (hectic type). The sepsis due to the jaundice gives rise to toxic symptoms.

3. **Physical Diagnosis.** The liver is moderately enlarged and tender. There is also a tender area in the region of the twelfth dorsal vertebra, 2 to 3 centimetres from the median line. (Boas.)

4. **Laboratory Diagnosis.** Leucocytosis is present. The serum reaction should be taken to determine the cause, and the blood examined to exclude malaria.

Differential Diagnosis. From *pyelophlebitis* the distinction is made by the absence of any cause for infection in the portal area; from *amæbic abscess* by the clinical antecedents, the physical signs, and the absence

of leucocytosis; from *malaria* by an examination of the blood; and from *simple cholelithiasis* by the absence of leucocytosis. (See Infections and Ball-valve Calculus.)

Cholecystitis.

Acute phlegmonous inflammation of the gall-bladder is attended by localized pain and tenderness, by high temperature, extreme prostration, and the rapid development of the typhoid state. Peritonitis rapidly ensues. It can be distinguished with difficulty from other forms of acute inflammation in the same region, unless there is a previous history of gallstones and tumor of the gall-bladder was present before the attack developed.

Suppurative inflammation of the gall-bladder may occur from gallstones and in infectious diseases. The colon bacillus, the diplococcus of pneumonia, and particularly the typhoid bacillus, give rise to infectious inflammation of the gall-bladder. The enlargement takes place suddenly and may increase, the tumor becoming tender and painful on palpation, while rigidity of the right rectus is marked. The direction of growth is toward the umbilicus. The general symptoms are those of suppuration. Hectic fever or markedly remittent fever occurs, and, unless surgical relief is given, peritonitis ensues from infection or from rupture. This complication may be suspected from the occurrence of collapse and increase of the local symptoms.

Either of these forms of cholecystitis is attended by pain in the region of the gall-bladder or in the epigastrium, or even as low down as the appendicular region. The pain is severe and paroxysmal. The symptoms of bacterial infection, of which vomiting and fever are the most prominent, rapidly follow. The general symptoms simulate those of appendicitis, intestinal obstruction, and pancreatitis.

The diagnosis is based on the :

1. **Clinical Course.** Primary cholecystitis follows acute gastroduodenitis and general infections; the secondary variety follows infections induced by foreign bodies and obstruction. The disease may occur at any age and in either sex. The mode of life appears to have little to do with its causation. The onset is variable; it may be fulminating, severe, or mild and gradual.

2. **Subjective Diagnosis.** The symptoms are pain from spasm or tension, and tenderness; nausea and vomiting.

3. **Objective Diagnosis.** The fever may be mild or severe, remittent or intermittent; chills occur in the fulminating type of the disease. Jaundice is not present.

4. **Physical Diagnosis.** Tumor at the end of the ninth rib exhibiting the characteristics of a swollen gall-bladder and movable with respiration unless inhibited by pain. There is spasm of the rectus muscle.

5. **Laboratory Diagnosis.** Leucocytosis is present in all infectious cases except those due to typhoid fever, and in 50 per cent. of the latter. It is important to take the serum reaction to determine the nature of the infection.

Differential Diagnosis. Cholecystitis simulates hyperæmia and infections of the liver, subdiaphragmatic abscess, diaphragmatic pleurisy,

pneumonia, pancreatic affections, perforation of a gastric duodenal ulcer, obstruction, uræmia, and endocarditis.

Cholecystitis in Typhoid Fever. The local infection may develop in the course of the disease—during the third or fourth week ; or in the period of convalescence. All grades are seen. It may or may not be accompanied by cholangitis. If it occurs in the course of the disease, its onset may be marked by an increase in the temperature, varying with the intensity of the local infection. In mild forms the fever may become a little higher than the continued range that had preceded. In severe forms it may become remittent or intermittent, and chills are not uncommon.

Nausea and vomiting are common in the beginning and may continue for several days. Pain in the region of the gall-bladder, and localized tenderness and rigidity of the right rectus muscle develop with the fever.

Fulminating cases may occur during the course of the disease, simulating perforation of the bowel.

The symptoms of onset are very characteristic. Pain is often so extreme as to simulate gallstones or lead to collapse. It is usually in the situation described, but may be referred to the epigastrium. The pain may seem to be general at first and even be marked at some distance from the gall-bladder, as in the right iliac fossa. Often the pain is diffuse during the first twenty-four hours and attended by general rigidity and distention. Frequently the local symptoms are overlooked, and I am convinced that many cases of so-called relapse in typhoid fever are due to mild cholecystitis. Leucocytosis is not necessarily present; the data concerning this point are quite insufficient. In one of my cases it was absent, although, following the law of leucocyte increase, such absence might have been due to the extreme infection.

Cholecystitis during convalescence from typhoid fever is not unusual. Contrary to Da Costa's experience, I have seen it quite as frequently at this period as at any other. Here, too, the cases may be mild, or fulminating with perforation. The onset in the post-febrile period takes place in the first or second week, when we are about to place the patient on solid food. In consequence, the change of diet is held responsible for the pain, tympany, and fever.

Confusion regarding secondary cholecystitis. Cholecystitis occurring in a subject who has gallstones or a displaced liver differs from primary cholecystitis in the local rather than in the general symptoms. The altered position of the gall-bladder changes the location of the pain and tumor. Thus, the tumor may be in the transverse umbilical, or in the median line. The tumor may be continuous with liver-dulness. An antecedent history of cholelithiasis aids in the diagnosis.

Differential Diagnosis. Cholecystitis can easily be recognized, the difficulty arising in distinguishing some forms of it from *appendicitis* being greatest, and yet these are cases of secondary cholecystitis, and therefore more obscure because due to displacement of the gall-bladder. Primary cholecystitis can be recognized by the clinical course, the physical signs, and the associate symptoms, together with the results of laboratory diagnosis.

In the distinction between the *catarrhal* and *suppurative* varieties we are deciding in part between mild and severe forms of infection. The degree of the infection is estimated by the severity of the local symptoms and the intensity of the toxæmia as indicated by the fever, the cardiovascular and cerebral symptoms, and the leucocytosis. An intense inflammation may arise without pus formation.

Cholecystitis must be distinguished from :

1. *Congestion of the Liver.* In cases of acute infectious endocarditis with cardiac dilatation, the left lobe of the liver often enlarges and is the seat of pain and great tenderness. This is accompanied by jaundice. An enlarged gall-bladder may be simulated, and as the general symptoms of infection prevail, that organ may be considered the site of primary infection. It is not unusual to have endocarditis without physical signs in the heart. Again, in a case of purulent pericarditis with congestion of the liver, the distention of the liver capsule was extreme, pain was excessive, and the picture was not unlike that of suppuration of the biliary passages.

2. *Syphilis of the Liver.* The history may help, but is often not to be relied on. Antecedent and associate conditions aid us here. The fever is commonly intermittent in syphilis. The paroxysms occur at fixed periods in the day however. Leucocytosis is absent. Justin's sign is not satisfactory. A localized tumor with the characteristics of an enlarged gall-bladder is usually not present. It may, however, exist, although it is not so tender. A rapidly growing localized gumma attended by fever and sweats may simulate gall-bladder infection. It has been the writer's experience to find a lymphocytosis more common in syphilis than in other infections.

3. *Multiple Abscess of the Liver or Pyelophlebitis. Portal Pyæmia.* The differential diagnosis is sometimes difficult or impossible. The antecedent history of abdominal infection is necessary to establish the diagnosis. The liver is large in multiple abscess; in cholecystitis, the gall-bladder. Pain is not marked in multiple abscess. Ascites, enlarged spleen, hæmatemesis, and diarrhœa are more common in portal obstruction.

4. *Abscess of the liver* due to amoeba is recognized by the history, by physical signs posteriorly rather than anteriorly, and often thoracic rather than abdominal, by the frequent absence of leucocytosis, the rarity of fever whose type is more or less slow, and by the presence of uniform enlargement or enlargement in one direction of the liver. The spleen is more likely to be enlarged in abscess than in non-typhoid cholecystitis. Abscess of the liver is neither so painful nor so acute an infection, and hence not so intense an infection as cholecystitis.

5. *Subdiaphragmatic Abscess.* The history, physical signs, and results of exploratory puncture are helpful in arriving at a diagnosis (see papers by Mason, Osler, and others). The condition manifests itself posteriorly, and the signs are abdominal as well as thoracic. The distinction from primary cholecystitis is not difficult.

6. *Diaphragmatic Pleurisy.* Diagnosis at times difficult. Exposure to cold is a feature in pleurisy.

7. *Pneumonia.* It is only necessary to call attention to the likelihood of confusing the infections in some cases. In pneumococcus infection with gastric and abdominal symptoms dominant, the pulse-respiration

ratio and the expiratory grunt may alone suggest the lesion, especially in children. When jaundice occurs early, the confusion may be increased.

8. *Pancreatic Diseases.* (See pages 1107–1113, Diseases of Pancreas.)

9. *Perforation of Gastric and Duodenal Ulcer.* The diagnosis is sometimes difficult, but the marked difference in the previous history is most helpful. Without such carefully worked out history the diagnosis may be impossible.

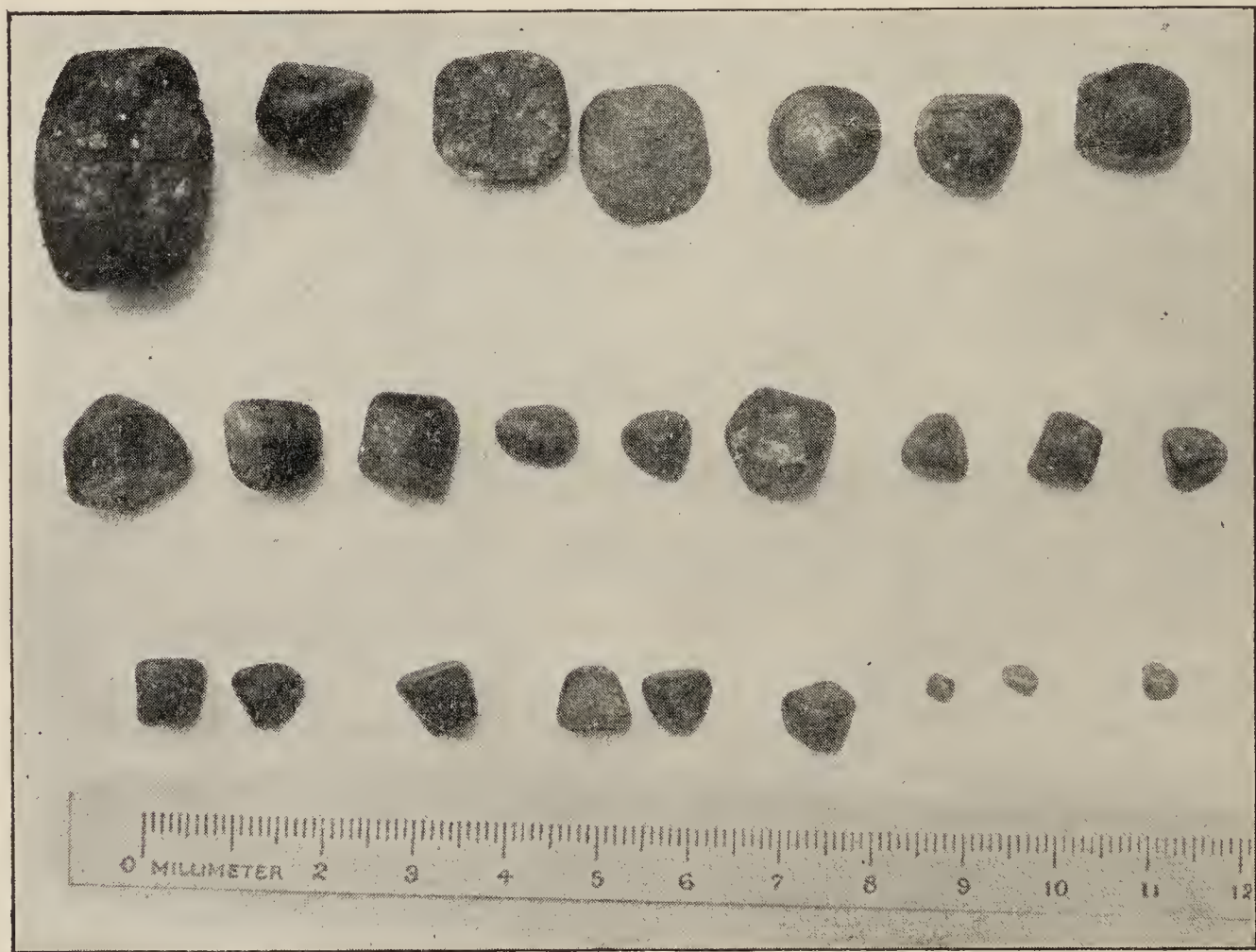
10. *Intestinal Obstruction.* This condition does not simulate hepatic and biliary disorders as frequently as it does pancreatic lesions.

11. *Uræmia.* Like hysteria, uræmia may simulate almost any other condition. In that form in which abdominal pain and vomiting are most prominent, the surgeon may easily be led astray, particularly as albumin and casts are almost always present in gall-bladder infections.

Gallstones.

Gallstones form in the biliary passages and may remain there without creating symptoms ; or the efforts to pass them may cause attacks of pain called *hepatic* or *biliary colic*, after which the stone may pass into the intestinal tract without causing further hepatic symptoms. It may become

FIG. 382.



Gallstones.

impacted in the biliary canal and set up catarrhal or suppurative inflammation, which in turn may be followed by stricture. Gallstones usually form, or at least show signs of their presence after the age of forty years, most frequently in women and in people who have led a sedentary life and partaken of rich and indigestible food. Individuals in different generations of the same family may be predisposed.

It is becoming increasingly well evidenced that gallstones are often the result of infection. This is particularly true of *typhoidal infection*. A history of typhoid fever preceding the development of gallstones is not uncommon, and typhoid bacilli have been known to furnish the nucleus about which they have developed. Typhoidal infection of the bile-passages acts by exciting catarrh of the mucous membrane; and in addition it is probable that the bacilli are agglutinated by the bile and thus afford a nucleus for the formation of calculi.

Hepatic Colic. The passage of gallstones may be attended by a slight amount of pain only, which, but for its location in the right upper quadrant, would pass for an attack of simple indigestion. In the large majority of cases the pain is severe. The attack may be preceded by biliousness or indigestion lasting twenty-four hours, and moderate pain or a sense of weight and fulness in the liver. It frequently follows the taking of food. Ringing in the ears, disturbance of vision, or undue flushings are said to precede it in some instances.

The attacks may be sudden. The patient is seized with *pain* along the margin of the ribs on the right side, or there may be pain above the ribs, over the liver, and in the right shoulder at the same time. From the hepatic region it extends to the median line. Very frequently the pain begins and continues in the epigastrium, or it may be most pronounced in this locality from the first. The pain is intense and paroxysmal. The patient is doubled up in agony. It causes more or less *collapse*. The pulse increases. *Vomiting* usually occurs at the same time, first of the contents of the stomach, and then of a yellowish, bile-stained fluid. The vomiting may be extreme so that the patient is tormented by the pain, the retching, and vomiting. The attack sometimes subsides as suddenly as it occurred, or wears off gradually. In very severe cases it is followed by symptoms of shock. The bowels are not disturbed during the attack. The urine may be suppressed; it is usually high-colored, and after the attack may contain bile.

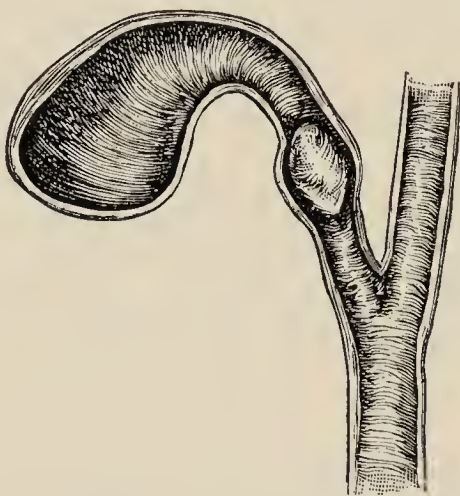
At the time of the attack there is considerable tenderness below the xiphoid cartilage and in the hepatic region. The *tenderness* is more marked on deep pressure in the gall-bladder region and to the right of the midclavicular line, at the margin of the ribs. The epigastrium may be slightly swollen. The tenderness persists after the attack, and the stomach may be weak or irritable for some time; pain, however, usually disappears at once. The attack may recur frequently until the stone has been passed, so that in twenty-four hours the patient may have a dozen or more paroxysms. After the attacks have subsided, light *jaundice* may supervene, but usually does not continue more than a week at most, during which period there are also symptoms of mild gastritis. (See Intestinal Colic.)

In some instances a *chill* precedes or immediately follows the pain, after which the temperature rises. After the paroxysm subsides, the fever disappears rapidly and is followed by profuse perspiration. If the gallstones have set up catarrhal inflammation, moderate fever may continue for a few days. (See Fever in Obstruction.)

During any paroxysm of hepatic colic it is desirable to determine

whether or not a gallstone has been passed. This can only be done by placing the feces in a sieve and pouring water upon them until they dissolve. Instead of gallstones, dark-colored granular bile, which has become inspissated, is sometimes seen in the stools. Bile in this form, according to Harley, gives rise to as much pain as true biliary concretions. If the stone is not passed, it may fall back into the gall-bladder and cause no further symptoms for a time, or become impacted in the ducts. The impaction may be such that no obstruction is caused by its position, the bile being forced through or around it; or complete obstruction may take place. (See Jaundice.)

FIG. 383.



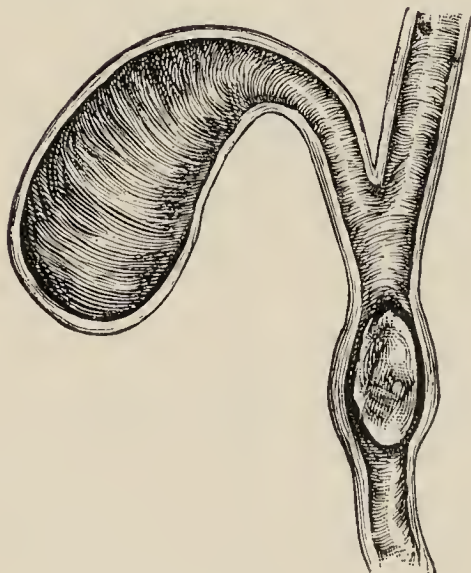
Gallstone in cystic duct.

FIG. 384.



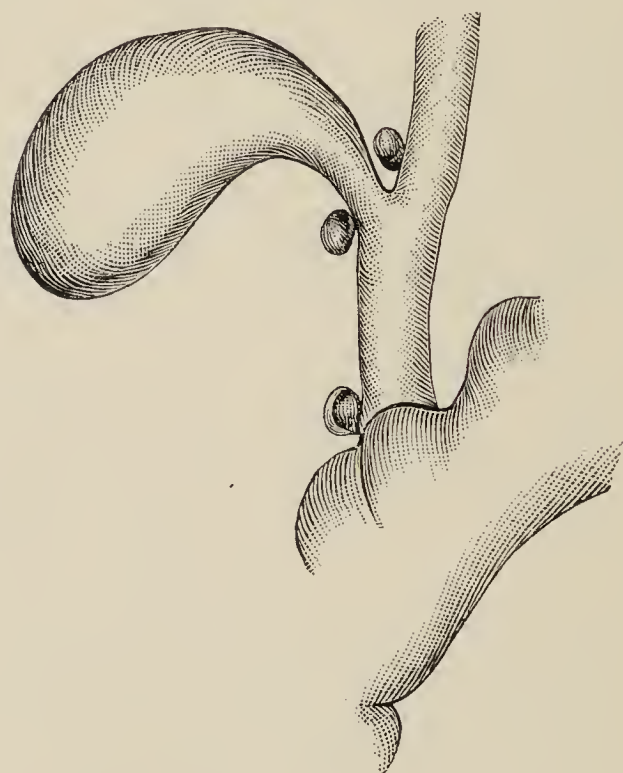
Gallstone in hepatic duct.

FIG. 385.



Gallstones in common duct.

FIG. 386.



Enlarged glands which cause jaundice.

Obstruction of the Common Duct by Gallstones. (a) In addition to jaundice paroxysms of chill, fever, and sweat occur, with catarrhal inflammation of the biliary passages. (1) The paroxysms resemble intermittent fever; (2) the jaundice may continue for years and deepen after each paroxysm; (3) hepatic colic may occur with the paroxysm; (4) the health fails but slightly. The paroxysms may occur daily or only once a

week, or they may be tertian and quartan in type. The pain is referred to other situations than the gall-bladder or the epigastrium. It is often relieved by vomiting or by certain positions of the body. The jaundice may be intermittent or remittent. On account of the obstruction in this situation the liver becomes enlarged. It is firm and smooth on palpation. The enlargement, as determined by percussion, is uniform. The gall-bladder is not enlarged. Fenger's thorough studies show that the *intermittent phenomena* are due to ball-valve action of a single stone. He also points out that emaciation is of common occurrence. (b) Gallstones may cause *suppurative inflammation of the ducts or of the gall-bladder*. The symptoms, both general and local, are severe. The fever may be intermittent, but is more likely to be remittent; jaundice is present, and is constant in degree. The local signs of enlargement and tenderness are made out. The patient dies of exhaustion or septicæmia. Sometimes the gall-bladder ruptures into the stomach or colon, and temporary abeyance of the symptoms results.

The Accidents of Gallstones. While these effects of the presence of stones in the biliary passages may rightly be considered as accidents, nevertheless their occurrence is so common as to be part and parcel of the history of gallstones. As accidents, we have most commonly the occurrence of localized peritonitis, which leads to dislocation of the gall-bladder, with constriction of the duodenum and secondary dilatation of the stomach; we also have the formation of biliary fistula, with passage of the gallstone into the contiguous organs or channels. The stone may ulcerate into the gall-bladder from one of the ducts, into the portal vein, or into the abdominal cavity—the most frequent accident. Perforation also takes place into the duodenum, into the colon, and, rarely, into the stomach. Such perforation can only be inferred from its secondary effects: (1) an attack of hepatic colic; (2) local inflammation with fever; (3) the occurrence of peritonitis, or the discharge of pus by the bowels or by vomiting. That it is due to gallstones is proved in those rare instances in which the stone is passed per rectum. Often the stone may be impacted in the intestinal canal, causing symptoms of acute obstruction; or in the rectum, causing local tormina and tenesmus. The perforation, however, occurs also in other directions. Sometimes a fistulous connection is formed between the gall-bladder and the urinary passages, calculi and pus being discharged in the urine. In other instances fistulæ between the bile-passages and the lungs are formed. The bile is coughed up and expectorated, sometimes with small calculi. In the most common form ulceration proceeds toward the surface, with formation of a cutaneous fistula. After the fistula has opened externally, gallstones in large numbers may be passed. If not, the cause of the fistula must be determined by the history and the results of probing, due attention being given to the condition of other organs.

Enlargement of the Gall-bladder.

Enlargement of the gall-bladder may be due to obstruction in the *cystic duct*. (Plate XLIV., Fig. 2.) The liver is not secondarily affected.

The enlargement is noted at the edge of the liver in the usual situation, and may gradually increase to an enormous extent, so that it has been mistaken for an ovarian cyst. The gall-bladder is often quite movable, and on account of its location and movability, as well as its elongated shape, has been mistaken for a floating or movable kidney. If the gall-bladder is not too large, it can be felt as a rounded or pyriform mass when the hand is placed along the margin of the liver, becoming more marked when the patient takes a full breath. The enlargement is not attended by any other symptoms except mechanical ones, unless the contents of the gall-bladder are purulent. In obstruction with simple enlargement the fluid obtained by aspiration is thin, of a mucoid nature, and alkaline in reaction. It may contain cholesterin-plates, and sometimes blood. It must be distinguished from the fluid of a hydatid cyst.

Diagnosis. *Simple* enlargement of the gall-bladder must be distinguished from enlargements due to inflammation.

FIG. 387.



Cancer of pylorus pressing on the cystic duct.

Enlargement of the gall-bladder, usually due to cystic obstruction as previously mentioned, may be mistaken for *floating kidney*, for *tumor of pylorus*, and for *ovarian cyst*. Tumors of the gall-bladder from any of these causes are recognized by their position and shape, and by the character of the tumor. The *position* varies. The tumor is usually situated in the gall-bladder region, but may extend as low as the groin, or may be so large as to distend the ribs and fill almost the entire abdominal cavity. If, however, the case has been under observation from the beginning, the tumor must have been found originally in the gall-bladder region. This region corresponds to the point where a line drawn from the acromion process of the right shoulder to the umbilicus intersects the costal margin. The tumor grows from this point toward the umbilicus in nearly all the cases. It can be recognized by its *shape*, which is pyri-

form, globular, or conical. The *character* of the tumor varies. It is usually tender and firm, but elastic on pressure, and movable. Fluctuation may often be detected. The septic gall-bladder is symmetrical and resistant to the touch. If the enlarged gall-bladder contains calculi, they may be felt as small hard masses, which transmit a grating sensation to the finger. On aspiration, if the cystic duct is obstructed, the mucoid fluid previously mentioned, or pus, is withdrawn. If the common duct is obstructed, bile will pass through the trocar.

The enlargement must be distinguished from *tumors of the liver, stomach, duodenum, pancreas, or lymphatic glands*. Tumors of the liver are usually due to *carcinoma*. They are multiple, associated with enlargement of the liver, with jaundice, ascites, enlargement of the spleen, and emaciation. Tumors of the *stomach, duodenum, and pancreas* are in a different position, and are attended by functional disturbance of the respective organs from which they spring. An *abscess of the liver*, if purulent, may simulate enlargement of the gall-bladder. If the abscess can be palpated, an area of induration is first felt, followed later by softening and fluctuation of the swelling. In judging of the nature of the tumor we must bear in mind the causes of abscess. In *hydatid disease* the tumor develops slowly; it is painless; it may yield fremitus, and, if movable, the course is slow and not attended by general symptoms. *Multilocular hydatid disease* can rarely be distinguished save by the difference in position of the tumor, which is nodulated, hard, and tender, and associated with jaundice, ascites, œdema of the legs, enlarged spleen, and great emaciation and prostration, with rapid decline. A *gumma* in the liver may occupy the region of the gall-bladder. It can usually be made out as continuous with the liver structure. It is tender and painful, but irregular; other signs of syphilis, or a history of the infection and of symptoms of a primary and secondary period will aid in the distinction of the disease.

Floating Kidney. The gall-bladder is larger and fixed at one end, whereas the entire kidney is movable. The gall-bladder may fluctuate, and there are symptoms of hepatic disease. On the other hand, the well-known symptoms of floating kidney, the shape of the tumor, the sensation of nausea induced by palpation, and association with gastropnoia and enteroptosis, point to the renal origin of the mass. *Tumors of the kidney*, such as sarcoma, hydronephrosis, and pyonephrosis, must be distinguished. 1. There may be changes in the urine. 2. In renal tumors the intestine is in front of some portion of them, or a zone of resonance is found between the liver-dulness and the tumor. 3. Renal tumors are fixed. They may, as in hydronephrosis, come and go, preceded by attacks of renal colic and attended by anuria. From *ovarian or uterine tumors* the diagnosis must be made by examination of the genital organs, although with the former there is often difficulty.

Enlargement on account of calculous obstruction must be distinguished from enlargement due to *cancer of the gall-bladder*. This is often difficult and can not be done without having the patient under observation for a long period of time. Cancer of the gall-bladder is usually *primary*. It may begin in the gall-ducts. In the larger number of cases it occurs in patients who have had gallstones. It is found

most frequently in females, and after the fiftieth year. Tight-lacing or pressure around the abdomen may predispose to it. The *symptoms* are pain, jaundice, emaciation, cachexia, and the presence of a tumor. The pain is localized and lancinating in character. Jaundice occurs in 70 per cent. of the cases and gradually increases in intensity. The tumor is situated in the gall-bladder region, to the right of the umbilicus. It is hard or firm, painful, and the seat of tenderness. The tumor is fixed. Sometimes the disease is found in the cystic duct, in which case the gall-bladder is enlarged. As history of gallstones is frequently obtained in both instances, it is impossible to distinguish the two forms of obstruction, save that in carcinoma the emaciation and cachexia may point to the true nature of the case. In tumor of the gall-bladder due to cancer the secondary effects on the liver are usually more marked than in tumor from other causes. The liver enlarges and its surface becomes irregular or nodular.

Diseases of the Portal Vein.

Disease of the portal vein, or occlusion of its branches in the liver, obstructs the flow of blood. The diseases of the portal vein are thrombosis, and adhesive and suppurative inflammation. Obstruction of the terminal venous radicles in the liver occurs in cirrhosis.

Thrombosis of the portal vein may attend cirrhosis of the liver, or occur secondarily to pressure upon the vein by a tumor. Disease of the pancreas was the cause of the pressure in a patient under my observation. As the result of thrombosis *adhesive inflammation* of the vein takes place, with or without the establishment of a collateral circulation to replace its function. The symptoms of disease of the trunk of the portal vein are the same as those of obstruction of the terminal branches, and are known as the symptoms of portal congestion. In one respect only do they differ. While we may have *ascites* in both; in thrombosis of the portal vein it occurs suddenly and the fluid rapidly accumulates again after tapping.

Suppurative inflammation of the portal vein is attended by symptoms resembling pyæmia, and is also called *portal pyæmia*. The inflammation is secondary, and depends upon inflammation somewhere in the portal area. It may follow appendicitis, infectious inflammation of the hemorrhoidal veins, or of the veins anywhere in the gastro-intestinal tract. Pus is carried into the liver by the portal current, and multiple hepatic abscesses are formed. Three pathological affections are therefore seen: (1) suppuration in the portal area; (2) inflammation of the portal vein; and (3) multiple abscesses of the liver (for the symptoms of which see Abscess).

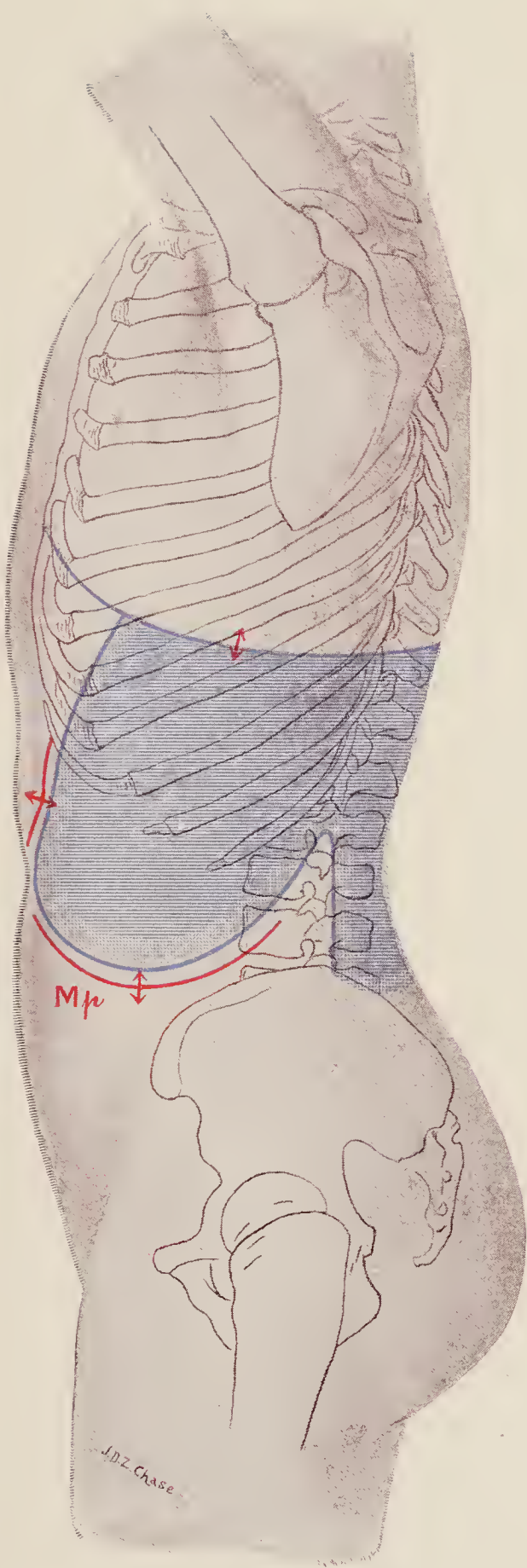
THE SPLEEN.

Enlargement of the Spleen.

Acute Enlargement. Enlargement of the spleen may be acute or chronic. Acute enlargement occurs in certain infectious diseases, particularly typhoid fever, typhus, smallpox, relapsing fever, scarlet fever, diphtheria, epidemic cerebrospinal meningitis, the malarial fevers and

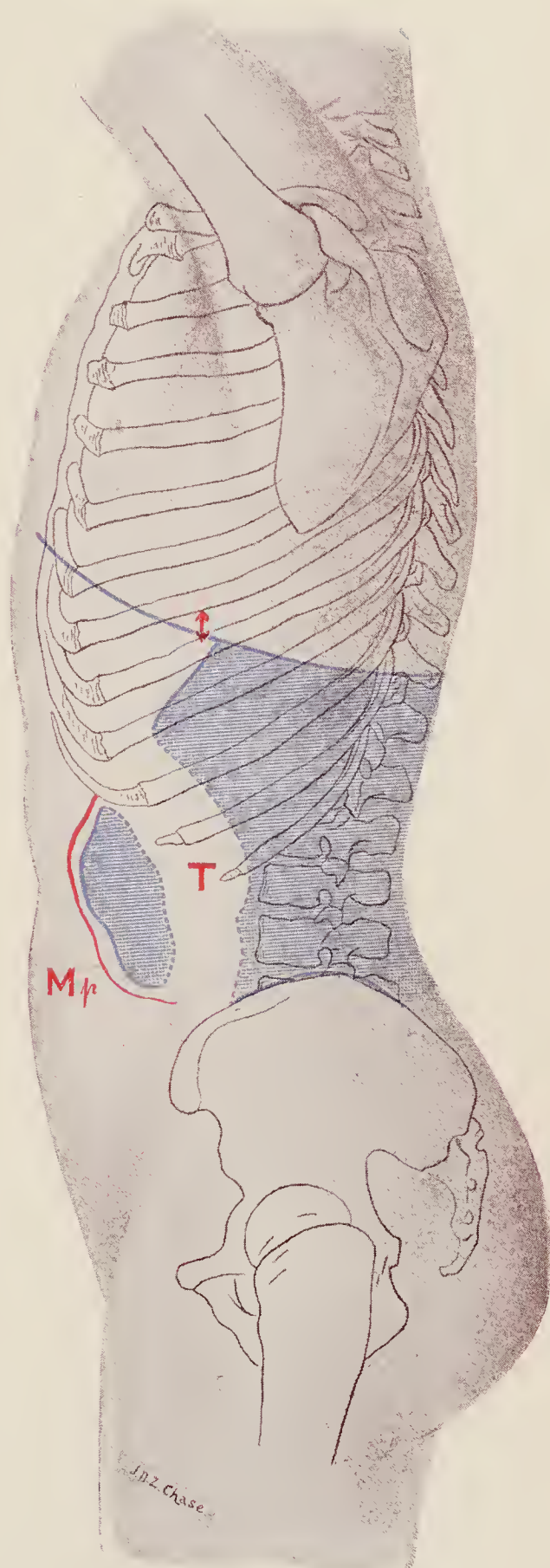
PLATE XLV.

FIG. 1.



Enlargement of the Spleen.

FIG. 2.



Tumor of the Left Kidney.

meningitis, and in diseases with blood-poisoning, as septicæmia, puerperal fever, and erysipelas.

A rare cause of enlargement is *acute splenitis*. Generally, it is the result of emboli lodged in the spleen and starting from an endocarditis. The area of splenic dulness extends rapidly, and there are local pain and tenderness on pressure, increased by coughing and deep inspiration; other symptoms are fever, nausea and vomiting, and occasionally delirium. If, as frequently happens in splenitis, emboli lodge in the kidneys also, the urine will be albuminous and bloody. If suppuration ensues, the fever becomes hectic, and the spleen continues to increase in size. Splenic abscess may, however, remain latent until rupture occurs.

Chronic Enlargement. Chronic enlargement of the spleen occurs as hypertrophy and as the result of amyloid disease, leukæmia and pseudo-leukæmia, chronic malarial poisoning (ague-cake), syphilis, hydatid tumor, and cancer. Enlargement is greatest in leukæmia, pseudoleukæmia, and chronic malaria. The spleen in well-marked cases of these affections may reach to the umbilicus and even beyond, filling up the hypogastrium and extending to the right iliac region; it may measure 13 or 14 inches in length and half as much in width, and be proportionately increased in thickness.

Primary splenic enlargement may occur without local or general symptoms; or anæmia, profuse hemorrhages, and brown pigmentation of the skin may be present at the same time; the hemorrhages are usually limited to the gastro-intestinal tract, and the anæmia is of a chlorotic type, with no change in the leucocytes. Splenic enlargement may also be associated with *cirrhosis of the liver* and *jaundice*, with gastro-intestinal hemorrhages and ascites. The affection is commonly known as *Banti's disease*. The blood-changes resemble those found in progressive pernicious anæmia, but differ in that nucleated red cells are absent or present only in small numbers, and the hæmoglobin shows relatively greater reduction. It may be confounded with chronic inflammation of the peritoneum, giving rise to ascites and associated with mediastinal pericarditis.

Diagnosis of Splenic Enlargement. (Plate XLV., Figs. 1 and 2.) Enlargement of the spleen can be distinguished from enlargement of the left *kidney* by the greater movability of the spleen. 1. The spleen does not extend so far back toward the spine as the kidney, so that the fingers can be thrust behind its posterior border, and if the other hand grasps the anterior edge, the organ can be moved backward and forward. Splenic dulness extends to the ninth rib or higher, while kidney-dulness has no thoracic area, but extends backward to the spine. 2. Again, the spleen is more movable with respiration than the kidney. 3. When the patient is in the knee-chest position, the spleen falls further toward the median line than the kidney. 4. An enlarged kidney has the colon in front of it, and hence its dulness is obscured by the tympany of the bowel. 5. The *shape* of an enlarged kidney is more globular than that of the spleen. The anterior surface of the latter is smooth and rounded, but at its junction with the flat posterior surface there is a sharp edge. 6. *Pain* in renal disease often shoots down the ureters and

into the testicles ; in diseases of the spleen the pain is generally localized to the splenic region, and may shoot into the left shoulder. 7. An examination of the urine will often make clear that the disease is renal, or a negative result will point to the splenic origin of the tumor.

Splenic enlargement is sometimes difficult to distinguish from *enlargement of the liver*, particularly of the left lobe. Careful palpation reveals the edge of the spleen, which descends further than the liver in full inspiration. After the anterior edge has been found, pressure with the other hand posteriorly will bring the spleen forward, which would not occur if the suspected enlargement were the left lobe of the liver.

Regarding the diagnosis of *splenic hypertrophy* (ague-cake) in *chronic malarial affections*, Osler says : "The history of malarial cachexia, the absence of lymphatic enlargement, and the blood-condition will usually be sufficient for the purpose of a diagnosis. Great increase in the white blood-corpuscles is not often seen in the chronic splenic tumor of malaria ; indeed, they may be much diminished in number. Toward the end in very chronic cases the clinical picture may be very similar ; the large abdomen, possibly ascites, dropsy of the feet, and irregular fever may resemble closely splenic leukæmia, and the absence of an increase in the colorless corpuscles may be the only marked difference."

Splenic Leukæmia. The diagnosis of splenic leukæmia (Plate XLVI., Fig. 1) rests principally upon the blood-condition, particularly upon the existence of a marked increase of white blood-cells, a large proportion of which are myelocytes. Red cells are decreased, and altered forms are present. In addition to characteristic blood-changes there is a great disposition to hemorrhages ; dropsies and priapism are common ; and, in later stages, fever, diarrhoea, great weakness, and grave complications such as pneumonia. Hemorrhage in splenic leukæmia occurs from the nose, bowel, stomach, gums, or kidney ; it may also be subcutaneous, inter-muscular, cerebral, or retinal.

Amyloid Spleen. Amyloid disease with enlargement of the organ, occurs in conditions of prolonged suppuration, especially when the bones are involved, and in chronic phthisis and syphilis. The spleen is enlarged, hard, and painless. The enlargement is rarely sufficiently great to produce distress on that account, and it is so commonly associated with a similar condition of the liver and kidneys, if not of other organs, that any constitutional symptoms produced by the spleen are apt to be masked by those produced by other organs.

Hydatid Disease. Hydatid tumor of the spleen rarely causes symptoms except when it becomes very large ; then it may give rise to discomfort and a dragging pain in the left hypochondrium. But hydatid tumors of the spleen are only exceptionally very large ; when large enough to admit of palpation, and when the tumor is situated anteriorly or projects from the lower border or from beneath the organ, the detection of fluctuation, the withdrawal of the characteristic fluid by aspiration, and possibly the presence of hydatid fremitus, will establish the diagnosis, when taken in connection with the gradual development of the tumor and exposure to possible infection. In the absence of physical signs of a cyst, the nature of the tumor can only be suspected from the habits of

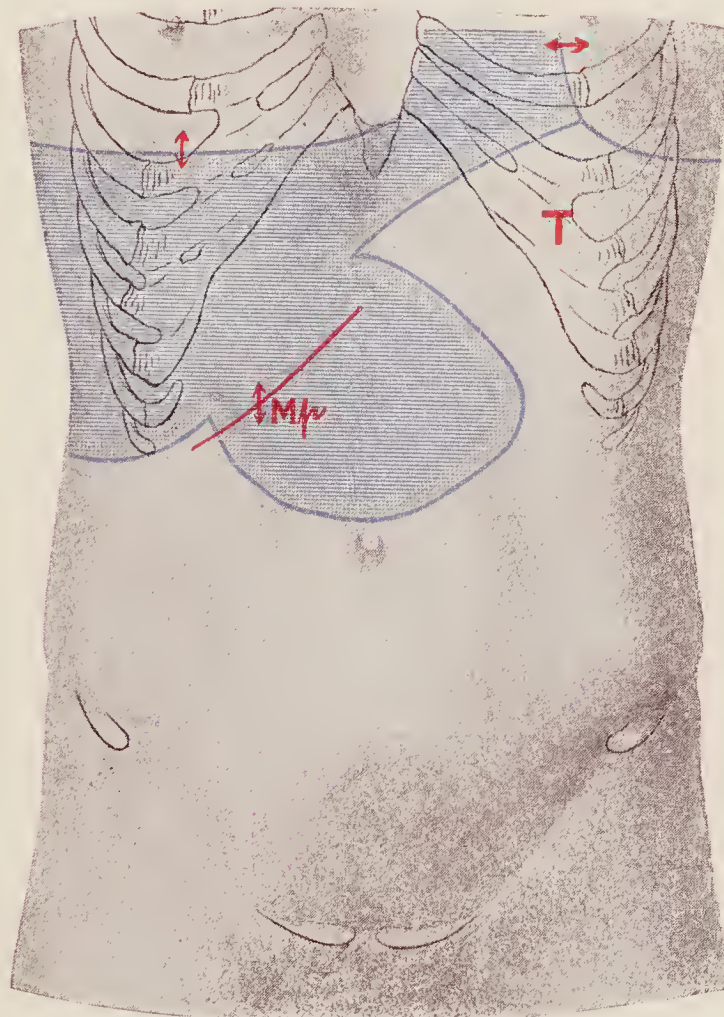
PLATE XLVI.

FIG. 1.



Leukæmia—Enlarged Liver and Spleen.

FIG. 2.



Cyst of the Pancreas.

the patient or his place of residence. Suppuration of the sac may be brought about by injury or rupture into the adjacent cavities, with grave if not fatal results.

Syphilis. *Inherited syphilis* and *chronic syphilis* are accompanied by enlargement of the spleen. They cause a chronic interstitial inflammation. The enlargement is usually not very great, and does not present characteristic features.

Malignant tumors of the spleen are rarely primary. The diagnosis must be made by noting malignant disease elsewhere, the very rapid enlargement of the spleen, with possibly nodules scattered over its surface, and the presence of cachexia and the usual constitutional signs of a malignant disease.

In *young children* enlargement of the spleen, with decided anæmia, is not uncommon. It is found associated most frequently with gastro-intestinal disease, rickets, syphilis, and malarial poisoning, and has been attributed to each of these diseases. In some cases none of these causes can be found, and the origin remains obscure. There is every reason for believing, however, that in most, if not all, instances the splenic enlargement is secondary to some other disease, usually an infection. Severe anæmia with splenic enlargement is uncommon in children over three years of age.

THE PANCREAS.

Just as the functional activity of the pancreas is separated with difficulty from that of other functionally related organs, so the aberration of such activity is discerned with the greatest difficulty. As the physiology and pathology are blended, so the symptoms are intermingled.

The pancreatic secretion aids in intestinal digestion, particularly in emulsifying fats; hence symptoms due to disturbance of this function are looked for, and it is, in a measure, true of all cases of pancreatic disease that there is some *intestinal indigestion*. For the purpose of determining whether the function of digestion of fats has been modified, the patient with suspected pancreatic disease is given fats in definite quantity, and the amount of fatty acids, soaps, and neutral fats in the stools is determined—a somewhat elaborate chemical procedure. If there is but little evidence of digestion of the fat taken—*i. e.*, if more than 75 per cent. of the fat in the stools is neutral fat—this is strong evidence of pancreatic disease, though the same conditions have in rare instances been seen in icterus without involvement of the pancreas or its duct. The mere observation of excess of fat in the stools, while it may arouse a suspicion of pancreatic disease, is of no real diagnostic value, as deficient fat absorption is most commonly due to absence of bile. The demonstration of good fat digestion, too, does not always show that the pancreas is normal; for it may be severely diseased without notable disturbance of fat-splitting ferment action. Sugar has been observed in the urine in many cases in which the pancreas was the principal seat of disease, and conversely, in many cases of diabetes morbid changes have been found in the pancreas. Both experiments upon animals and observation of

human beings show that the pancreas is closely connected with the origin of diabetes in some cases. Glycosuria, however, is not constant in pancreatic lesions. The three symptoms—*intestinal indigestion*, *fatty stools*, and *glycosuria*—are therefore not diagnostic of pancreatic disease, but only afford presumptive evidence of its presence.

Tumor of the Pancreas.

The most striking symptoms of disease of the pancreas, apart from those due to the morbid process, suppuration, or cancer, are those due to the pressure of the *tumor* upon surrounding structures. It may press upon the gall-duct and cause jaundice. From its situation in the epigastric region it may resemble an aneurism, or a tumor of the pylorus or of the transverse colon. Tumors of the pancreas are usually due to *cancer*. This is usually of the scirrhus variety, and generally primary. The enlargement cannot be distinctly made out unless the patient is very much emaciated. When it has advanced considerably, it may simulate aneurism, but is distinguished by the difference in the character of the pulsation. In an aneurism the pulsation is distensile; in disease of the pancreas it is an up-and-down movement—the hand is lifted with each pulsation of the aorta. Tumor of the pylorus is excluded largely by the more superficial position of the mass, its association with pyloric obstruction, and the absence of jaundice, which occurs less frequently than in disease of the pancreas. A pyloric tumor is more movable and may change its position after the stomach has been inflated with gas or distended with fluid. Examination with the patient on the hands and knees may aid in the distinction between the two. A tumor of the transverse colon is recognized by its nearness to the surface, its movability, its association with more or less constipation, and the occurrence of intestinal hemorrhage.

The general symptoms of the cancerous cachexia; the occurrence of intestinal indigestion, or loss of fat digestion; the gradual onset of jaundice; the occurrence of deep-seated epigastric pain; an immovable tumor, with glycosuria, make a symptom-group very characteristic of cancer of the pancreas. When the patient is on a milk diet, an examination of the feces will show that there is deficient pancreatic digestion with loss or reduction of the fat-splitting action.

Hemorrhage into the Pancreas.

We owe to F. W. Draper and Prince the greater part of our knowledge of hemorrhage into the pancreas. Since they have published the results of their labors the affection has been frequently recognized. The attack comes on suddenly in perfect health, and usually terminates life in a short period. It is associated with arteriosclerosis, particularly of the splenic artery and its branches, and the form associated with inflammation is probably frequently an infection. Nothing in the occupation or conduct of the patient at the time appears to favor the development of hemorrhage, except the occurrence of trauma. He is seized with *severe pain*, localized in the upper part of the abdomen. It increases in severity, and

may be intermittent like colic. *Nausea* and *vomiting* take place almost at the same time. The vomiting becomes obstinate. Extreme depression rapidly sets in, and the patient becomes anxious and restless. Collapse ensues in a short time. The extremities become cold and the forehead is covered with sweat. The pulse increases in frequency, and rapidly diminishes in strength. It soon becomes imperceptible. The pain and vomiting call attention to the upper abdomen. It is tender on pressure; the tenderness may extend throughout the entire upper half of the abdomen. Tympanites may develop. There is constipation in many cases. The temperature remains *normal*, or becomes *subnormal*. The pain, the vomiting, the anxious and restless state continue without relief.

From the above group of symptoms it can readily be seen that the *diagnosis* is obscure. The disease may be mistaken for perforation of the stomach by ulcer, although the vomiting is not, as a rule, so persistent and frequent. Intestinal obstruction in the upper portion of the tract presents allied symptoms. The hemorrhage symptoms, however, are more pronounced in pancreatic hemorrhage. Pallor of the face is sure to ensue. The vomiting is not fecal in character. Constipation can be relieved. It is, however, difficult and in many cases impossible to establish a diagnosis of hemorrhage into the pancreas. The rapidity of development of the symptoms is of importance. The pain and collapse of the rupture of an aneurism of the aorta simulate *pancreatic hemorrhage*.

Acute Hemorrhagic Pancreatitis.

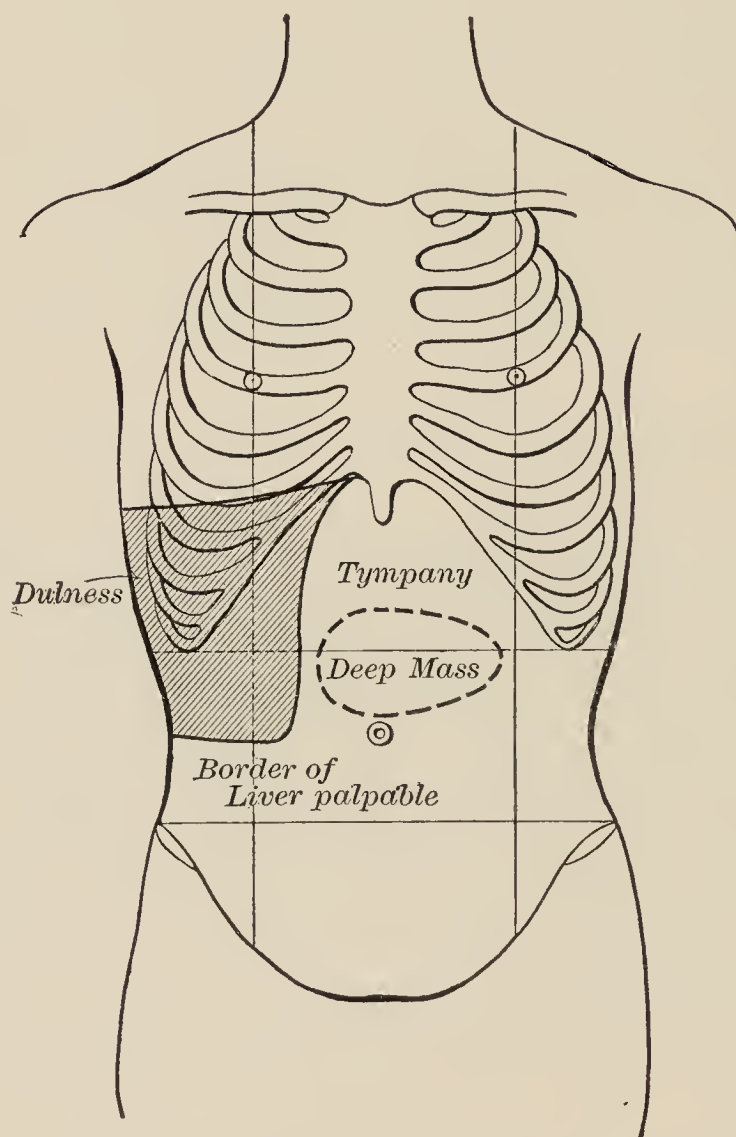
For our knowledge of this disease we are largely indebted to Fitz. He collated the facts from the literature, and, adding the results of his own valuable observations, has made the symptomatology sufficiently clear to allow of the occasional recognition of the affection during life. It usually occurs after the middle period of life, although it may occur in early childhood, the youngest patient known to the writer being eight months of age. It is more common in males, in those addicted to alcohol, and in fat subjects. The patient has often been the subject of attacks of indigestion, or of epigastric pain, or biliary colic. Indeed, cholelithiasis and infectious cholecystitis are well-known causes. It followed typhoid cholecystitis in one of my cases, injury in another, and the cause was unknown in another. A blow on the abdomen or injury in the lumbar region appears to have been the exciting cause in a number of cases.

The *attack* develops suddenly, not unlike pancreatic hemorrhage—indeed, the two are very closely related, the inflammation being often the result of hemorrhage into the organ. There is *violent pain*, which is at first complained of in the upper abdomen, although it is sometimes general. *Nausea* and *vomiting* are present in all the cases, *constipation* in most of them. The abdomen is frequently the seat of tympanitic distention. In many instances an obscure *tumor* can be made out in the lower epigastric region. *Collapse-symptoms* supervene; although *fever* may occur, the temperature rising to 102° F. The cases terminate by the fourth day, sometimes even earlier. The pain and collapse are probably due to pressure of the effused blood upon the celiac plexus; while

the fever and the appearances of intoxication are probably the result of infection. Violent delirium resembling acute mania and not unlike that seen in atropine-poisoning, occurs in some instances. Symptoms of localized peritonitis arise, and if the patient lives, the tumor increases to a considerable size.

Diagnosis. The symptoms resemble those of acute *intestinal obstruction*, *intoxication with irritant poison*, or *perforation* of the gastro-intestinal or biliary tract, or an attack of hepatic colic. In several instances laparotomy has been performed for the relief of supposed obstruction. The intense pain in the epigastrium, with violent vomiting and distention of the upper abdomen, without a possible cause for obstruction, are favorable to acute pancreatitis. The difficulty of diagnosis, however, is so great that resort

FIG. 388.



Tumor of the pancreas.

to laparotomy is justifiable in order to determine the exact nature of the condition. In a most interesting case reported by W. S. Thayer, the diagnosis of acute pancreatitis (confirmed by laparotomy) was based upon the history of previous attacks of pancreatic pain, with fever, vomiting, and collapse, occurring in an adult who was over-fat and an alcoholic; the exclusion of disease in other organs and the absence of a history of gallstones, gastric ulcer, or abscess from other causes; the occurrence of pain; the presence of a deep-seated tumor with indistinct signs of fluctuation, which was not movable with respiration, and the dulness of which was

not continuous with, or of the same character as that of adjacent solid organs. Epigastric tympany was also a point in favor of pancreatic disease. The accompanying figure indicates the site of the tumor in Thayer's case.

Suppurative Pancreatitis.

Suppurative pancreatitis occurs in adults under forty years, more frequently in males. Symptoms continue during several weeks, and many persist for a year. *Pain in the epigastrium* is complained of, associated with *irregular vomiting*, the latter persisting in spite of care as to feeding. *Fever* is irregular in type, and exhaustion ensues. Jaundice, fatty diarrhœa, and glycosuria have been observed in some cases. In a case under my observation obstruction of the portal vein took place and was followed by ascites, which was large and recurred rapidly after tapping. In this patient pain and gastric disturbance were absent. There was no fever. Emaciation, constipation, and a tumor above the umbilicus were present; the emaciation was extreme. The tumor was ill-defined, painless, apparently superficial. Many other symptoms of pancreatic disease pointed out by Roberts were present, such as apathy and despondency and bronzing of the face. The man was forty-two years old and addicted to the use of alcohol. He was thought to have cirrhosis of the liver. As happened in this case, the pus may accumulate in the duodenojejunal fossa and fill up the cavity of the lesser peritoneum, with more pronounced symptoms of tumor than occur in similar fluid accumulations in the above-mentioned cavity.

Gangrenous Pancreatitis.

This may follow later upon hemorrhage into the pancreas. The symptoms are extremely obscure. *Collapse* may occur, following *pain*, which is of longer duration than in the acute form, or *vomiting*, which is not so persistent. A patient of mine, upward of sixty years old, suffering from dyspepsia, vomited blood in the course of an illness which was characterized by loss of flesh and weakness. The anæmia became very profound after the gastric hemorrhage, and exhaustion was extreme. There was no marked tumor, but only resistance in the region below the xiphoid. There were dulness and tubular breathing at the base of the left lung. Fever was absent. Death ensued from exhaustion. A small flat carcinoma was found at the pyloric end of the stomach, but there was no perforation. Gangrenous pancreatitis, with signs of an ante-mortem hemorrhage, were found. The accumulation had taken place behind the stomach and colon, but in front of the kidney; its outer wall was bounded by the spleen. It was limited above by the diaphragm. Pleuritis and small pulmonary abscesses at the base of the left lung were found.

In some instances the pancreas has sloughed into the bowel; and in two such cases recovery took place after its discharge from the rectum.

Chronic pancreatitis is not recognized during life, although its possible

presence must be considered in all cases of diabetes, and in jaundice not otherwise explained.

Cyst of the Pancreas.

The cysts are not really true cysts, but accumulations of pancreatic fluid in the lesser peritoneal cavity. (Plate XLVI., Fig. 2.)

Causes. A pancreatic cyst may result from obstruction of the pancreatic duct with impacted biliary calculi; the obstruction is sometimes at the orifice. In some instances there is a history of a previous attack of so-called biliary colic or colicky pains in the upper abdomen, with vomiting but without jaundice—a condition characteristic of the presence of calculi in the pancreatic ducts. The disease occurs in adults, and often follows a trauma, a blow in the epigastrium being a frequent exciting cause.

Symptoms. The symptoms are those of a tumor occupying the upper abdomen in the median line or a little to the left, in the left upper quadrant. There is a sense of weight and fulness in the epigastrium. The tumor may be sufficiently large to fill the entire abdominal cavity and simulate ovarian tumor. As a rule, the cyst *grows slowly*, but in some cases the growth is quite rapid. *Fatty diarrhœa* is not present. In contrast to cancer, *pain is absent*. In this, as in other diseases of the pancreas, *diabetes* may be present. Senn has called attention to the peculiar complexion of patients suffering from pancreatic cyst; he describes it as of an unhealthy yellow, dirty, or earthy hue. The writer also considers that the history of the case, the location of the tumor and its relation to other organs are factors to be reckoned with in the diagnosis of pancreatic cyst.

Physical Signs. The objective signs are those of a *tumor* to the left of the median line, encroaching upon the left lobe of the liver above and extending almost to the transverse umbilical line. The cyst may *move with respiration*. At first it is always found in the region occupied by the pancreas, its exact position depending somewhat upon the portion of the pancreas from which it originated; it may be below the right lobe of the liver, below the xiphoid, or in the *left upper quadrant*; in the great majority of cases it occupies the last situation. Körte in a series of sixteen cases observed that the greatest prominence of the mass was *below the navel*. The tumor is smooth and may fluctuate; it is not hard and lobulated. On account of its presence the diaphragm may be arched so that the heart is dislocated upward and to the left; the apex is found in the third interspace. It displaces the stomach forward and to the right, the transverse colon downward, the diaphragm and the contents of the chest upward. The cyst may be movable in respiration. It also causes increased dulness behind on the left side, the upper border approaching the angle of the scapula. *Exploratory puncture* in either instance determines the nature of the fluid, and may establish the diagnosis. Boas does not think the chemical character of the fluid is sufficiently typical to establish a diagnosis. (See Examination of Cystic Fluid, page 696.)

Diagnosis. Pancreatic cyst must be distinguished from cancer of the

pancreas or adjacent organs, aneurism, hydatid cyst of the liver, the spleen, or the peritoneum, affections of the retroperitoneal glands, hydro-nephrosis, cystic disease of the suprarenal capsule, circumscribed peritonitis with exudation, ascites, and cystic disease of the ovary. *Pain* is an important symptom of disease of the pancreas in its more acute manifestations ; it must be distinguished from the pain of intestinal obstruction and the pain of perforative peritonitis. The pain is always localized in the region below the xiphoid, or, in general, is confined to the upper half of the abdomen. It exactly simulates the pain of the affections just described. This resemblance is more pronounced because of the association of vomiting and collapse in obstruction and perforative peritonitis. Pain, although not so intense, but of a colicky nature, attended by diarrhœa or constipation, in some instances with intestinal hemorrhage, may be due to *calculous disease of the pancreas*. Frequently this form of pain can be recognized if other symptoms of pancreatic disease, such as glycosuria, steatorrhœa, and intestinal obstruction are present.

CHAPTER XI.

DISEASES OF THE KIDNEYS.

THE kidneys are affected by disease from several sources: (1) the great vascular supply is subject to the alteration which takes place in any large arterial area, either from direct *hyperæmia*, through the influence of the vasomotor nerves (see *Hyperæmia*), or from passive hyperæmia or *congestion* through the central organ of the circulation. (2) The blood-vessels may be the seat of *thrombosis* or *embolism*, particularly the latter, causing renal infarction. (3) *Infectious* material, as micro-organisms or toxins, is carried to the kidney, and in passing through the structure gives rise to *inflammation* either of an infective or of an irritative character. Similarly, *poisons* that are ingested, and products of metabolism excite irritation and lead to inflammatory changes.

But the kidney is open to attack from sources lower down in the urinary tract. Through the bladder and ureter *infection* may extend upward, causing the consecutive inflammatory processes which are often seen after disease of the urethra, bladder, or ureter. The kidney is at the apex of a system of tubes or channels. Any alteration of them, whether mechanical or functional, has a secondary effect upon the kidney. Obstruction of the ureter or obstruction of the conduits beyond leads to consecutive hypertrophy, inflammation, and atrophy. (See *Morbid Processes*.) If the urine is abnormal, one of these three causal conditions may be present.

Symptoms due to the Morbid Processes.

The morbid processes that may take place in the kidney are such as are common to all organs—congestion, inflammation, degeneration, morbid growths, and hydatid disease, and the symptoms are such as accompany similar processes elsewhere. The *general* symptoms of the morbid processes, chiefly fever and emaciation, are not marked except in the case of infectious inflammation or of morbid growths, as carcinoma. *Fever* occurs in acute nephritis, perinephric abscess, suppurative and tuberculous nephritis, pyelitis, and with twists of the ureter, in floating kidney. *Emaciation* occurs in chronic, suppurative, and tuberculous nephritis and carcinoma. Other general symptoms in renal disease are due to the interference with the function of the organs which usually results. *Pain* is the only *local* symptom due to the morbid process; *swelling* is the only physical sign.

Anatomical and Physiological Symptoms.

The symptoms of renal disease are also due to the functional or anatomical alteration of the kidney. But the structure is so closely inter-

woven with the function that morbid changes in one imply morbid changes in the other. As the anatomical alterations are usually beyond the pale of physical investigation, we find that functional symptoms alone are apparent. Hence, we look for *changes in the urine* and for symptoms resulting from abeyance or cessation of the renal function. Rarely we have *enlargement* due to obstruction of the channels causing hydro-nephrosis.

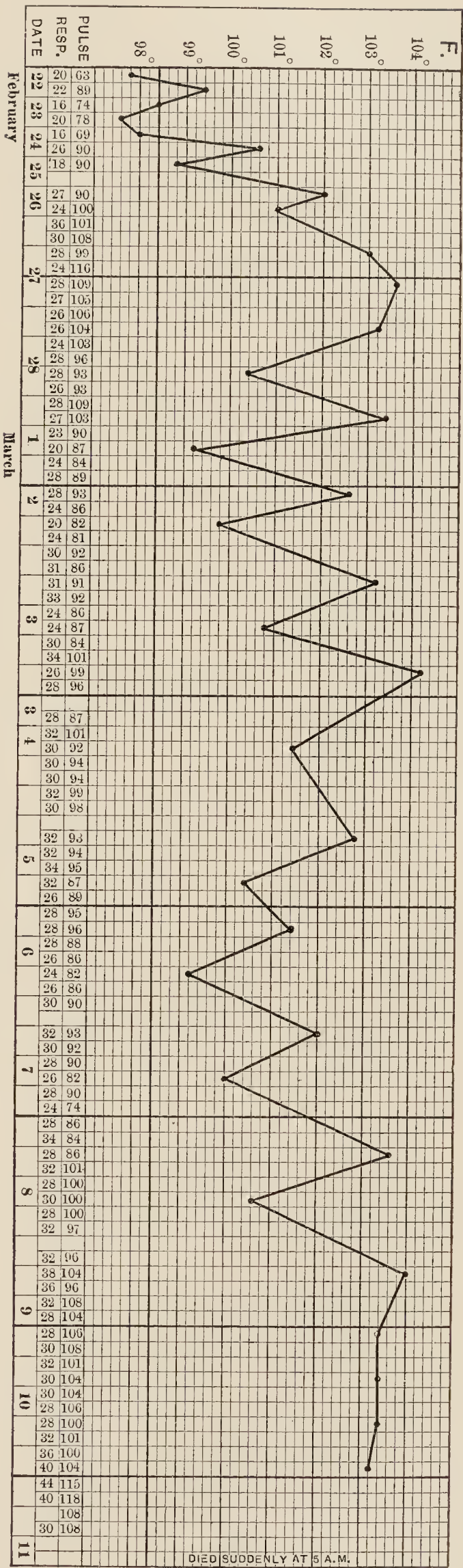
The symptoms due to alteration of function are: 1. *Uræmia*. 2. *Cardiovascular symptoms*, including *dropsy* and *anæmia*. 3. *Alterations of the urine*. 4. *Disturbances of micturition*. The symptoms of renal disease are therefore both subjective and objective.

Diagnosis of Renal Diseases.

The recognition of renal diseases is based upon evidence of the history, the subjective diagnosis, the objective diagnosis, the physical examination, and the laboratory diagnosis.

Historical Diagnosis. Age, sex, and habits are important factors. Acute nephritis attends any age, but chronic nephritis occurs late in life and with the onset of cardiovascular degenerations. The habits and hygienic conditions causal in the latter are likewise induced in the former. A previous history of infections, notably scarlet fever, is usual in acute and chronic renal processes. Chronic intoxications, as with metals, are also followed by renal inflam-

Temperature in uræmia simulating typhoid fever.



Subjective Diagnosis. The chief symptoms which demand inquiry are pain and disturbed micturition.

Pain. (See Chapter VIII., Part I.)

Disturbances of Micturition. There are four causes of frequent micturition : (1) disease of the kidneys, the ureters, or the bladder ; (2) an increase in the amount of urine, as in diabetes ; (3) concentration of the urine, as in fevers, gout, or acute nephritis ; (4) reflex irritation or pure neurosis.

Increased frequency of micturition occurs in almost all organic affections of the genito-urinary system. It is seen in all forms of congestion and inflammation of the kidneys. In some forms of nephritis the increased frequency may be due to increase in the amount of urine as well as to increased sensitiveness of the organs. In chronic nephritis it may not be noticed save that the patient is called upon to pass urine at night, being aroused from sleep for the purpose. Disease of the ureter and disease of the bladder are also associated with this troublesome symptom. It occurs in its most aggravated and characteristic form in renal colic due to calculus, or when any foreign substance is located in the ureter or bladder. The frequency amounts to six, eight, or even a dozen times in an hour. It is often associated with tenesmus, the patient having a constant desire to urinate, but passing only a small quantity at a time. This form of tenesmus is more frequent in renal calculus and when the bladder or urethra is the seat of disease.

Objective Diagnosis. Uræmia. Under symptoms due to impairment of the functions of the kidney belong the various manifestations of uræmia. Diseased kidneys do not eliminate the products of tissue-waste, which are poisonous materials. The toxic matter is retained within the blood and produces toxæmia which may be acute or chronic. In *acute uræmia* the manifestations develop suddenly and continue but a short period of time, with alarmingly active symptoms, until death or recovery. In *chronic uræmia* the onset is gradual. The manifestations may be limited to one or two conditions, as headache or morning nausea, or they may include the more pronounced symptoms of uræmia.

Latent uræmia was first recognized by Sir William Roberts. It is seen in its most characteristic form in calculous suppression. The patient for several days will have subnormal temperature, myosis, occasional vomiting, and toward the end twitching of the voluntary muscles, and slight drowsiness. At the end of five or ten days coma, convulsions, or dyspnoea ensues.

Nervous Symptoms. Headache. The pain is situated in the occipital region and may extend down the neck. It is severe and of a bursting character, and may be associated with giddiness. In both acute and chronic nephritis headache is often the first manifestation ; it may be associated with eye symptoms ; it may be present on waking, and continue only through the morning hours. In acute uræmia it persists throughout the attack. Numbness and tingling of the fingers are often complained of at the same time.

Delirium. The delirium may be mild. This is usually the case in the typhoid state or if a subnormal temperature prevails. It is sometimes attended by delusions. There are often subsultus and picking at

the bedclothes. The delirium may amount to true mania, and the patient may exhibit other maniacal symptoms. On the other hand, the patient may be noisy, restless, and sleepless. Melancholia and delusional insanity may develop after the violent nervous symptoms of uræmia have passed off.

Convulsions. A convulsion may be the first indication of disease of the kidneys, or it may succeed a few days of persistent headache, or follow an attack of uræmic vomiting. The convulsion resembles epilepsy, and hence is known as an epileptiform convulsion. If the spasms recur in rapid succession, the interval is occupied by delirium or coma. If they are infrequent, the patient's mind may be clear in the intervals. Sometimes a focal or Jacksonian epilepsy occurs instead of the true epileptiform convulsion. The *temperature* is usually elevated. In worn-out subjects, or in those who are debilitated from exhaustive diarrhœa or some other cause, the temperature may be subnormal. A temporary blindness often follows the convulsion (*uræmic amaurosis*). Uræmic deafness may occur.

Coma. After the convulsion consciousness may return, or the patient may lapse into stupor, followed by complete coma. Coma may develop without convulsions, or immediately succeed a general convulsion. Headache or eye symptoms may precede the coma. In some instances the patient lapses into a typhoid state, in which the tongue is heavily furred and the breath very offensive. Unless the coma is profound, there is usually some twitching of the muscles of the hands and face.

Local Palsies. Dercum was among the first to call attention to the occurrence of *uræmic monoplegia* or *hemiplegia*. The cases resemble central cerebral disease. The nature of the palsy is inferred from the results of the examination of the urine and the condition of the heart and arteries. Palsy develops suddenly and may occur after a convulsion.

Cramps in the muscles of the calves, particularly at night, are of common occurrence, and should always lead to an examination of the urine.

Pruritus, local or general, is another nervous symptom that may be of uræmic origin.

Pain in the upper abdomen, particularly in the median line, is a frequent precursor of more severe uræmic symptoms. It is of uræmic origin itself. It may be seated in either of the upper quadrants, and thence extend to the lower portion of the abdomen.

Respiratory Symptoms. *Uræmic Dyspnœa.* Modifications of the breathing often accompany symptoms of uræmia. The dyspnœa may be constant or it may occur in paroxysms, or the two types may alternate. A common type in the uræmia of chronic nephritis is the Cheyne-Stokes breathing. Paroxysmal dyspnœa usually occurs at night, and resembles asthma in every respect. Cheyne-Stokes breathing continues even through the period of coma, although it is not necessarily associated with it. (See page 99.)

In addition to dyspnœa, the occurrence of *inflammatory pulmonary complications* may be the first indication that the condition of the urine should be inquired into. Bronchitis, pneumonia, and pleurisy are common complications of uræmia.

Gastro-intestinal Symptoms. *Loss of appetite* is common. It is attended with absolute distaste for food after a small portion has been taken. *Nausea* may be continuous, or more frequently limited to the early morning. *Vomiting* may be paroxysmal, occurring chiefly in the early morning; or it may be sudden in onset, uncontrollable, and continue until nervous symptoms of uræmia develop. Urea is found in the vomitus. The matter ejected is profuse, of low specific gravity, and at first acid in reaction. In cases of chronic vomiting the vomitus may be alkaline. The odor is often sufficient to cause its recognition. *Constipation* is the rule in the course of chronic Bright's disease. One of the manifestations of uræmia is *diarrhœa*, often in the form of sudden profuse serous purging. This may be so extreme as to cause collapse, or may usher in coma and convulsions. *Hiccough*, although a muscular affection, is usually associated with gastric disturbances. Fermentative dyspepsia, gastralgia, chronic gastritis, enteritis, and ulcerative colitis are of common occurrence.

Cardiovascular Symptoms. These symptoms are due to the retention of morbid products. The poison which is not excreted circulates through the system. One of its effects is irritation of the vasomotor nerves of the bloodvessels. Excitation of these nerves causes peripheral contraction of the smaller vessels. At once the flow of blood is obstructed, so that, on account of the contraction, hypertrophy of the heart rapidly ensues. The first prominent symptom, therefore, is due to changes in the heart muscle.

Hypertrophy of the Heart. The most pronounced change is *hypertrophy*. The persistent spasm of the peripheral vessels causes increased arterial tension. The blood-pressure is raised and causes increased accentuation of the aortic second sound. *High tension* in the artery is recognized by the peculiar character of the pulse and by means of the sphygmograph.

Dilatation of the Heart. Unfortunately, hypertrophy of the heart can not always be kept up. If it fails, we then have a second condition of the heart which is frequently found in renal inflammations; it is dilatation. The state of the coronary arteries predisposes to this condition of the heart muscle. The previously mentioned arterial tension favors the development of chronic endarteritis with general atheroma, and the coronary arteries take part in this process. The endarteritis hinders cardiac nutrition, dilatation of the heart muscle follows, and later comes the development of two other conditions, atrophy and myocarditis.

Here may be mentioned other relations of the heart and kidneys: (a) We have renal disease following forms of cardiac disease. In dilatation of the heart passive congestion of the particular organ takes place. The kidney quickly becomes the seat of such congestion. In the course of simple dilatation or of valvular heart disease, the secondary dilatation, passive congestion, and chronic inflammation develop slowly. Embolism may also occur. (b) Renal disease and cardiac disease may develop at the same time from a common cause, as alcoholism, gout, or endarteritis.

In addition to high arterial tension and accentuation of the aortic second sound, the objective symptoms of atheroma of the aorta and arteries are associated with chronic inflammation of the kidney. (See Endarteritis.) It is important to bear in mind that the two conditions frequently occur together, and also that in all instances of arterial disease

the condition of the urine must be looked into. It is needless to say that symptoms due to rupture of a bloodvessel, particularly in the brain, or to aneurism may supervene in the course of renal inflammation.

Hemorrhages. The arteries are very liable to rupture, causing epistaxis, retinal hemorrhage, hemorrhages from the bowels and lungs, and hemorrhages underneath the skin (petechiæ). Frequent, copious hemorrhages from any portion of the body should call attention to the condition of the urine.

Ophthalmoscopic Changes. The eye-ground should always be examined ; indeed, the patient himself by his complaints often directs attention only to the eye, the examination of which discloses the presence of an albuminuric retinitis. The changes may occur in the acute or chronic forms of nephritis, although they are more common in the latter. 1. A diffuse, slight opacity and swelling of the retina, due to œdema. 2. White spots or patches of various sizes, for the most part the result of degenerative processes. 3. Hemorrhages. 4. Inflammation of the intra-ocular end of the optic nerve. 5. Atrophy of the retina and nerve may sometimes result from and succeed the inflammatory changes. These changes may affect one eye only (Gowers). It must not be forgotten that temporary blindness may occur independently of retinitis.

Dropsy. Dropsy may occur in all forms of nephritis. It is most common in the acute varieties, but it is also present in chronic diffuse nephritis. Renal dropsy usually begins in the face. It may develop suddenly in acute forms, and in grave diseases of the kidneys œdema of the eyelids may continue for a long time. All varieties of dropsy, from local œdema to extreme anasarca, are observed. The serous cavities are also filled. The œdema is usually associated with a diminution of the quantity of urine, and its subsidence is attended by increased diuresis. In chronic disease dropsy is usually due to dilatation of the heart. (See page 950.)

Anæmia. Anæmia is a frequent symptom in all forms of nephritis ; it is usually marked. It is associated with the peculiar pallor of kidney disease, and attended by all the other usual symptoms of an impoverished state of the blood.

General Symptoms. The cause of renal disease, so far as symptoms pointing to the kidneys are concerned, is often latent. Instead of renal symptoms a generally depraved state of the system with *emaciation* and *weakness* may be the only indication of disease. Lassitude without cause demands an examination of the urine.

Appearance of the Face and Cutaneous Symptoms. In inflammatory affections of the kidney the appearance of the face and the condition of the skin are often characteristic and point at once to an examination of the urine. The face is pallid and of an ivory whiteness ; in chronic renal disease the pallor gives way to an ashen-gray or sallow tint, and the skin becomes dry and harsh. In rare instances the skin is covered with a powdery substance resembling hoar-frost, which is derived from urea and is analogous to the chalky deposits of urates known as tophi.

Physical Diagnosis of Enlargement of the Kidney. Renal Tumor.

Enlargements of the kidney may be detected by percussion ; the width of the kidney is increased, and the percussion-dulness therefore extends further to the right or left, according as the right or left kidney is affected. As the causes which produce enlargements of the kidney sufficiently great to be detected by percussion do not, with rare exceptions, involve both kidneys at the same time, comparison of the two sides is of great value in the diagnosis.

Renal tumors give rise to swelling in the loin with muscle resistance if there is inflammation. They may extend forward, and are then palpable between the ribs and the transverse umbilical line. The mass is never notched, is usually smooth, and often takes the shape of the kidney if that organ is involved in its entirety. Otherwise the outline is not reniform. The bowel is usually in front of the mass, although in tumors of the right kidney the cæcum and colon may be pushed to the inner side, and in tumors of the left kidney the colon may be pressed outward.

The diseases of the kidney attended by enlargement are : *malignant tumors, cystic kidney, hydronephrosis and pyonephrosis, abscess, and perinephric abscess.*

Laboratory Diagnosis. Alterations of the Urine. (See Chapter XLIII.)

Congestions of the Kidney.

Acute Congestion of the Kidneys. Any diseased kidney may become the seat of acute (or active) congestion. The insult of a prolonged surgical operation and extirpation of one kidney may be followed by acute congestion ; but the commonest cause is some irritant poison. Death may take place from uræmia. *Urine* : The quantity may be diminished or there may be total suppression ; catheterization is often required. The urine contains albumin in considerable quantities, blood, and numerous epithelial casts.

Chronic congestion of the kidneys is usually a part of general venous stasis—hence also spoken of as passive congestion ; valvular heart disease with secondary dilatation, and pulmonary emphysema often give rise to the condition, which is not at all uncommon. *Urine* : the quantity is diminished, the color dark, the specific gravity high—1020 to 1030. Uric acid and urates are present in excess. Albumin is not present at first, but soon makes its appearance at intervals and in small quantities, later becoming constant and more abundant. The sediment contains hyaline casts and a few red blood-cells.

The condition is recognized by its association with congestion in other organs ; by the diminution in the amount of urine, its high specific gravity, and excess of uric acid and urates. This form of congestion is serious because it leads to chronic nephritis.

Degenerations of the Kidneys.

Degeneration is always a secondary process and may be either acute or chronic. The primary causes are : inorganic poisons, as arsenic or phos-

phorus; the toxins of the infectious diseases; chronic visceral disease; and disturbances of the circulation.

In *acute degeneration* of the kidneys the quantity of urine may be normal or diminished; albumin is present in varying quantities, and the sediment contains casts and red blood-cells. The urinary changes may be the only symptoms, or signs of uræmia may develop at once. Dropsy and cardiac hypertrophy do not occur.

Chronic degeneration of the kidneys may be a sequel of chronic congestion, or may be produced by alcoholism or syphilis. It may develop during the course of gout or malarial cachexia, and is a not infrequent event in pulmonary phthisis and when there is a chronic suppurative process going on anywhere in the body. *Symptoms*: The simpler forms do not produce clinical symptoms. Loss of flesh and strength, anæmia, and in rare cases the development of the typhoid state mark the course of the severer forms of degeneration. In uncomplicated cases of degeneration there is no hypertrophy of the left ventricle, and albuminuric retinitis is rare. *Urine*: The findings are variable. The urine may be abundant, scanty, or suppressed; the specific gravity is normal, but albumin and casts are present.

Amyloid degeneration of the kidneys is associated with the same process in other organs, and the *symptoms*, when any are present, are due to the anæmia and cachexia of the primary disease and to the involvement of the other viscera. According to Purdy, dyspepsia and diarrhœal attacks are common. The liver and spleen are usually enlarged; œdema is not present as a rule, and uræmia is a rare occurrence. *Urine*: The quantity may be normal, diminished, or increased; it varies from time to time in the same case, being influenced by complicating conditions such as diarrhœa; as a rule the quantity is more abundant than in health. The color is very pale; the specific gravity ranges between 1008 and 1014. Albuminuria of considerable degree is a constant symptom, and hyaline casts and white blood-cells are always found; the presence of other casts points to a complicating nephritis. The distinctive features of the casts found in amyloid disease are their large size and waxy appearance. The *diagnosis* of amyloid disease is based upon the presence of a cause, the urinary changes, and signs of similar disease in other organs.

Inflammations.

The best classification from the pathological point of view of the inflammations of the kidney is that given below; but this classification does not always fit the clinical picture.

Acute Nephritis. *a. Acute Tubal or Parenchymatous Nephritis.* This is essentially an acute degeneration of the tubular epithelium, with little or no involvement of the glomeruli. The cells of the tubules are cloudy, swollen, granular, often fatty and vacuolated. Congestion is a marked feature in this variety, which is the type of mild kidney-lesion occurring in the course of typhoid and scarlet fever, diphtheria, pneumonia, and the other infectious febrile diseases, and is produced by the

action of the specific toxins. It is often classified, and perhaps more properly, as *cloudy swelling* or *parenchymatous degeneration*.

b. Acute Exudative Nephritis. In addition to the degenerative changes of the preceding form (*a*), there is an exudation of serum, fibrin, or red blood-corpuscles into the capsule of Bowman and into the tubules. When the exudation of red blood-corpuscles is very great, the term *acute hemorrhagic nephritis* is employed.

c. Acute Glomerular Nephritis. This form is characterized by a proliferation either of the cells of Bowman's capsule—*acute capsular glomerulonephritis*—with pressure-changes in the glomerular tufts; or of the cells of the glomerular capillaries—*acute intracapillary glomerulonephritis*. The glomerular lesion explains the oliguria or anuria frequently observed in this type of the disease. The lesions characteristic of (*a*) and (*b*) may and usually do accompany the glomerular changes. This is the type of nephritis commonly seen during convalescence from scarlet fever, and often spoken of as *scarlatinal nephritis*.

d. Acute Interstitial (non-suppurative) Nephritis. This unusual form occurs practically only in connection with diphtheria and scarlet fever. The characteristic pathological alteration consists in an infiltration of lymphoid and plasma cells between the tubules, in severe cases obliterating entire groups of tubules. Extensive degeneration of those structures results, with practically no change in the glomeruli. No distinctive urinary changes have been described.

e. Acute Interstitial (suppurative) Nephritis or Abscess of the Kidney. For the sake of completeness this form should be added. (See page 1120.)

The ætiology of all the forms of acute nephritis is practically the same. The form (*d*), which is practically always a complication of diphtheria or scarlet fever, differs from the others in that the urine is scanty, high-colored, and of high specific gravity; but the number of observations on the urine in this type of nephritis is small.

Chronic Diffuse Nephritis. **a. Large White Kidney.** This is often described as *chronic parenchymatous nephritis* in contradistinction to the chronic interstitial or contracted kidney; the term *chronic tubular nephritis* has also been employed. The kidney substance shows a slight, uniformly distributed increase of connective tissue; fatty or hyaline degeneration of the tubular epithelium; and, occasionally, a small accumulation of lymphoid cells. The glomerular tufts are lobulated and smaller than in normal specimens; sometimes there is a slight accumulation of serum in the capsule of Bowman. The condition may lead to the small white kidney.

b. Amyloid Kidney. This form is usually included under the degenerations (*q. v.*).

c. Small Red Kidney. Clinically, this form is known as the kidney of *chronic interstitial nephritis* or as the contracted, granular, gouty, or arteriosclerotic kidney; the last-named variety differs from the others only by its association with arteriosclerosis. The histological changes are: over-growth of connective tissue, atrophy and fibroid changes in the glomeruli, atrophy of some of the tubules with dilatation of others, and

an infiltration of lymphoid cells. In the arteriosclerotic form the characteristic lesions are found in the vessels.

Acute Tubal or Parenchymatous Nephritis (Cloudy Swelling). This form of nephritis is characterized by congestion, exudation of plasma, transudation of red and white blood-cells, and changes in the epithelium.

Causes. There may be a history of exposure to cold, but the disease occurs at times without definite cause. It is a frequent complication or sequel of the infectious diseases, notably scarlet fever, and is the expression of a peculiar type of typhoid fever. The kidney of pregnancy is one of the manifestations of this form of nephritis; the course of diphtheria, erysipelas, pneumonia, and septicæmia is frequently marked by its development; it may complicate dysentery and acute tuberculosis; and it is one of the modes of termination of diabetes.

Symptoms. The course of the disease may be mild, presenting only the urinary changes, or there may be, in addition, local and general symptoms.

In mild cases the *urine* is diminished in amount; micturition is frequent; the color is dark, the specific gravity usually high. There is a small amount of albumin, with a few epithelial and blood-casts, and sometimes blood. At the termination of the disease hyaline casts are found.

In severe cases the disease is ushered in by *chill*, attended and followed by *pain* in the loins, with *fever*, *headache*, and restlessness. There are a large amount of albumin and an abundance of hyaline, granular, epithelial, and blood-casts. Free red and white blood-cells and epithelium from the pelvis and tubules are found. The fever continues; the pain in the loins is sometimes very severe, and, unless the urine is examined, may be mistaken for lumbago. Within the first forty-eight hours the characteristic symptoms that follow the chill and accompany the urinary changes are headache, sleeplessness, more or less profound stupor, muscular twitchings, and general convulsions. Eye symptoms may be present. Instead of cerebral symptoms there may be marked dyspnoea; and with both, nausea and vomiting are of common occurrence. The heart's action is increased in force and frequency. The left ventricle rapidly becomes hypertrophied. The aortic second sound is accentuated. The *pulse* is hard and exhibits the characteristic features of high tension. From the onset of the first symptom, or within the first week, two other striking phenomena arise, *dropsy* and *anæmia*.

Dropsy or oedema is one of the most constant symptoms. It appears first in the face, especially the eyelids, and may be limited to this region, being most marked in the morning. From the face, in severe cases, it extends to the lower extremities and to the scrotum, and thence over all the body. *Anasarca* is the name applied to general dropsy in which the connective tissue is infiltrated with serum; it is recognized by the pallor of the swollen surface, the pitting on pressure, and the absence of heat and pain. (See page 228.)

Effusion may take place into the serous cavities, the pleura, pericardium, or peritoneum. In some instances there is oedema of the mucous membranes, as the conjunctiva, the soft palate, and the glottis.

Dyspnœa may be a pronounced symptom, being due to uræmia (uræmic asthma), œdema of the glottis, effusion into the pleura, or bronchitis. If dilatation of the heart occurs, the ensuing dyspnœa may be a direct result or due to the secondary œdema of the lungs.

With or without the occurrence of nausea or vomiting there is always *loss of appetite*, and the bowels are usually *constipated*.

The *fever* is usually moderate and irregular in type. *Prostration* is common; often there is emaciation. Symptoms of *uræmia* may occur at any time.

Exudative nephritis with excessive *pus* formation is of sudden onset, characterized by high fever and extreme prostration. There are rapid *emaciation* and the early development of the *typhoid state*, which is preceded by delirium, headache, and stupor, with great restlessness. In this form there is but little, if any, dropsy. Large numbers of red and white blood-cells and the usual casts are found in the urine. There is not so much diminution in the quantity of the urine as is usually seen. The disease may arise without apparent cause, or complicate scarlet fever or diphtheria. This form is very fatal, and resembles *acute meningitis*, from which it is diagnosticated by the changes in the urine.

Acute Glomerular Nephritis. In this form there are an over-growth of connective tissue and an excessive growth of the capsule-cells in the glomeruli, in addition to the lesions of the first form. The whole kidney is not necessarily affected, but only portions at a time. *Symptoms:* The onset is sudden. The subjective symptoms previously described are present in a marked degree. *Nervous* symptoms (uræmia) are most pronounced. *Dropsy* develops rapidly and to an extreme degree. There is rapid *loss of flesh with anæmia*. The remaining symptoms tally with those of the first affection.

The *urine* is scanty, bloody, and of high specific gravity. The microscopical appearances are like those of acute exudative nephritis. If convalescence is established, the urine becomes more abundant, with a corresponding fall in the specific gravity. The albumin and casts may persist for a time, but eventually disappear.

The *diagnosis* of the various forms of acute nephritis is based upon the examination of the urine. *Ætiological* associations are of value. The more pronounced cases follow scarlet fever and pregnancy. In the latter condition the course is usually slow. There may be no symptoms until the onset of uræmia. In some instances the disease resembles typhoid fever. In cases in which the onset is sudden, with early uræmic symptoms, it must be distinguished from epilepsy, delirium, and mania.

Chronic Productive or Diffuse Nephritis (Large White Kidney). In chronic inflammations the formation of new tissue always takes place. *Exudation* practically does not occur. The essential difference in the various forms lies in the amount of connective tissue. In this form the amount is slight and the degenerative changes in the tubules very prominent. In the contracted form there is great increase of the connective tissue; sclerosis of glomeruli is its predominating characteristic.

Causes. The diffuse form usually follows acute productive nephritis and chronic congestion or degenerations of the kidney. It develops in the

course of syphilis, tuberculosis, endocarditis, disease of the bone, and prolonged suppuration. Frequent exposure to cold and wet, residence in damp dwellings, and the alcoholic habit are causal conditions. It usually occurs in middle life, more frequently in men; but as a primary disease usually attacks young adults.

Symptoms. The disease develops slowly. General symptoms may be first observed. *Dropsy* may develop at first and continue throughout the disease, or recur at long intervals. The appearance of the patient is striking. The skin is of a peculiar *pallor* and pasty in appearance. The scleræ are very white. The *anæmia* which gives rise to the pallor is profound, and often closely resembles that of pernicious anæmia. The anæmia is due to diminution in the hæmoglobin and reduction in the number of red blood-cells.

Headache and *sleeplessness* are common symptoms. Pronounced acute uræmia does not often occur. *Chronic uræmia* may prove fatal by the patient lapsing into a typhoid state in which delirium alternates with stupor.

Urine. It must not be forgotten that the course of the disease and the urinary symptoms are often quite variable in chronic nephritis. The urine may be normal in amount, but during the exacerbations it is scanty or suppressed. The specific gravity and the amount of urea lessen. In the most rapid cases it varies between 1012 and 1020. In chronic cases it falls as low as 1005 and even 1001. In the later stages the amount of the urine and the specific gravity may both be increased. Albumin is present in large amounts. When the disease is most active and the dropsy at its height, the quantity of albumin is very large. In the quiescent period of the disease the amount is lessened. Epithelial, fatty, and granular casts are abundant; red blood-cells are often found.

Albuminuric retinitis frequently develops in the course of the disease.

Dyspnœa is often observed, and may be due to any one of the many causes previously described as producing this symptom in the course of nephritis. It is frequently limited to sudden attacks which develop in the night or early morning. There is often some bronchial catarrh.

Nausea and *vomiting* are common symptoms. The appetite is lost.

Hypertrophy of the left ventricle takes place in all cases, except in those persons who had been previously weakened by other disease. The right ventricle is often hypertrophied also. The second aortic sound is accented, and the pulse is of high tension. Symptoms such as *headache* and *vertigo* arise on account of the profound *anæmia*.

Course. Delafield has well outlined the course. The constant symptoms are anæmia, dropsy, and albuminuria. 1. The symptoms may be continuous and progressive in severity, death taking place at the end of one or two years on account of dropsy or uræmia. 2. The symptoms may continue for several months and the patient finally improve. Recurrent attacks take place, the symptoms being more severe with each attack. In the intervals of the attacks there is a small amount of albumin in the urine. 3. The patient may apparently recover, but the urine continues to be of low specific gravity, and contains some albumin. A fatal attack of uræmia, or an apoplexy, or the onset of an acute disease may cause an

exacerbation of the renal symptoms. 4. The symptoms may persist in a mild degree for years, the patient at the same time feeling comparatively well. 5. Spasmodic dyspnœa may be the first and only symptom for a long time.

The disease is characterized by remissions and exacerbations. During the exacerbations any one of the prominent symptoms that occur in renal inflammations may be present. Œdema is the one symptom which occurs most frequently, and is likely to continue the longest. The disease lasts from three months to three years, and may pass into the second variety of chronic inflammation.

Chronic Interstitial Nephritis. This form of nephritis is called also *chronic, non-productive, diffuse nephritis, granular kidney, or cirrhosis of the kidney*.

The kidneys are diminished in size, the capsules are adherent, and the surface is roughened. There is an over-growth of connective tissue with atrophy of the epithelium and of the tubules, and dilatation of some of the tubes, forming cysts.

Causes. This form of nephritis follows chronic congestion of the kidney, and is also caused by alcohol, lead, gout, syphilis, malaria, and by chronic endarteritis. The last condition, as well as cirrhosis of the liver and pulmonary emphysema, frequently develops hand-in-hand with the nephritis. This form of nephritis is notably prevalent in several generations of different families, so that a hereditary history is often readily obtained.

Symptoms. The onset of the disease usually occurs late in life, although well-defined cases may occur as early as the twenty-fifth year. The progress at first is very insidious, and the disease may have advanced to an extreme stage without the occurrence of a single symptom. Death, indeed, may be due to other causes; or a person in apparently perfect health may suddenly manifest symptoms of uræmia, or may develop apoplexy or some other usual accompaniment of interstitial nephritis. With the exception of the state of the urine, the only symptom present may be the loss of flesh and strength. At the same time the skin becomes dry and harsh. Œdema, however, is not usually present unless there is dilatation of the heart. Special symptoms are due to uræmia, to changes in the heart and arteries, and to neuroretinitis.

URÆMIC SYMPTOMS. These symptoms may occur at any time in the course of the disease. Headache is most common and constant. It may occur early in the morning only, or continue throughout the day. It may be continuous and cause sleeplessness. General neuralgic pains may be present instead of severe headache. Muscular twitchings or general convulsions may be other pronounced symptoms, or, instead, delirium, mild or violent, stupor, and coma may come on. These symptoms occur suddenly or develop very gradually. In acute uræmia with the above-mentioned cerebral symptoms there is peripheral spasm of the arteries, causing high arterial tension, and there is elevation of the temperature. The fever may rise to 103° or 104° F., but is usually about 102°, and is irregularly continuous. After the patient lapses into deep coma, if the attack is fatal, the tension of the pulse is lost, and it is increased in

frequency and diminished in strength. In chronic uræmia the cerebral symptoms develop gradually. The temperature is likely to be subnormal, particularly if diarrhœa or other debilitating influence is coincident. The pulse is rapid and feeble.

PULMONARY SYMPTOMS due to uræmia are quite common. They may be the first expression of uræmia. This is seen in all forms of nephritis. The most marked symptom is dyspnœa, which is spasmodic and of short duration. The attacks may occur frequently, and are usually increased by exertion and aggravated by a recumbent posture. The shortness of breath may occur in the early morning hours, or may continue throughout the day.

Pulmonary symptoms other than those due to uræmia may be caused by an intercurrent bronchitis, pneumonia, or pleurisy. Chronic bronchitis or œdema of the lungs may be present on account of dilatation of the right heart. The chief pulmonary symptoms that point to these conditions are dyspnœa and cough.

Spasmodic dyspnœa is the first and sometimes the only symptom for a long time. Later the renal symptoms become pronounced, pointing to the true nature of the disease.

GASTRO-INTESTINAL SYMPTOMS. Catarrhal gastritis almost always complicates nephritis. In addition, gastric symptoms due to uræmia, and hence to deficient action of the kidney, ensue. The most common is the occurrence of morning nausea or of morning vomiting; the occurrence of spasmodic vomiting at irregular periods, or the occurrence of violent, acute vomiting, which is followed in two or three days by other symptoms of uræmia. The bowels are usually constipated. When the disease is complicated with cirrhosis of the liver, intestinal catarrh is common, and intestinal ulceration with consequent diarrhœa is frequently found. The onset of uræmia may be characterized by violent and profuse serous purging, which of itself may cause collapse and death.

NEURORETINITIS is a frequent complication of nephritis, and may advance more rapidly than other complications, so that dimness of vision, blindness, or other eye symptoms may cause the patient to consult an oculist before attention is called to the condition of the kidneys. The occurrence of this complication points at once to the necessity for an examination of the urine.

VASCULAR SYMPTOMS. It is common in the course of an interstitial nephritis to have accidents due to the condition of the arteries that accompanies this disease.

On account of the atheroma, and aided by the hypertrophied heart, rupture of the vessels frequently takes place. Apoplexy is, therefore, of common occurrence, and hemorrhage into other organs sometimes occurs.

The renal disease is often not suspected until after the patient has had an attack of apoplexy. The course of this form of nephritis is varied very much by the occurrence of complications, notably emphysema, endocarditis, or cirrhosis of the liver.

CATARRHS. There is always a tendency to chronic inflammations of the mucous membranes and to acute inflammations of serous membranes in the course of chronic diffuse nephritis. It is necessary, therefore,

when local inflammations of this character are present, to make thorough and repeated examinations of the urine, especially in a patient over forty years of age, with a history of one of the causal factors previously mentioned.

URINE. The quantity is increased, the color clear, and the specific gravity low. The albumin is small in amount, or may be absent. Repeated examinations extending over a considerable period of time may disclose its presence. Hyaline casts are present in small numbers. In some cases it may be necessary to examine a dozen or fifteen slides before they are found. Sometimes there are a few red blood-cells. Rarely the urine is bloody at irregular periods in the course of the disease, or actual hæmaturia may take place.

HEART. The left ventricle hypertrophies. The aortic second sound is accentuated. The pulse is of high tension. The arteries become more prominent and present all the signs of endarteritis. In the later stages, as nutrition fails, dilatation of the heart takes place, with regurgitation at the mitral valve and the development of a train of symptoms due to these changes. Among others we find general malaise, palpitation of the heart, dyspnœa, œdema, and visceral congestions.

Course. Several clinical forms of interstitial nephritis are observed. In the latent form the disease may have advanced to an extreme degree without any symptoms of renal disease during life, death taking place from an intercurrent disease or accident. On the other hand, palpitation of the heart may be the only symptom complained of, and the observer finds a hard pulse, general atheroma, and hypertrophy of the left ventricle with accentuation of the second sound. Apart from this the patient may enjoy very good health. The danger lies in the occurrence of pneumonia or inflammation of a serous membrane. Often the local inflammatory symptoms are slight or masked by the symptoms of renal disease, which develop rapidly.

In another group of cases some special symptom only may be complained of. In some instances it may be gastric catarrh, in some eye symptoms alone may be present, while in others hemicrania or other forms of headache are observed. With the headache there is usually vomiting. Again, we may have constant neuralgia or persistent muscular rheumatism as the only symptom. Nose-bleed is a symptom which may be the only indication of chronic nephritis, particularly if the epistaxis occurs frequently.

In other cases the course is not latent, but characterized by a series of attacks at varying intervals.

During the attacks the symptoms resemble those of the acute form of nephritis, with acute uræmia, the occurrence of dyspnœa, loss of appetite, nausea, and vomiting. The tension of the arteries is higher at the time of the attacks. The urine contains albumin, and is of low specific gravity during the time of the attack; during the interval albumin is found at irregular times.

Suppurative Nephritis (Abscess of the Kidney). Infectious matter is conveyed to the kidney either through the *blood*, as in pyæmia and ulcerative endocarditis (rarely dysentery and actinomycosis), or by the *ureters*,

as when it follows pyelitis or cystitis. A wound may infect the kidney directly.

Symptoms. The symptoms are those of the primary disease, and the affection is usually recognized only post mortem. Or the symptoms are merely those of suppuration. Pus is seen in the urine only on rupture of the abscess into the pelvis of the kidney.

Tuberculous Nephritis. Fever, emaciation, anæmia, and prostration characterize the course of the disease. Tuberculosis is usually found elsewhere. There may be no other symptoms. Sometimes hydronephrosis is present. A tumor is often present. It may be in the loins, or may be in front, above, and a few inches to the right or left of the umbilicus. The *urine* is normal or contains pus and detritus, or even bacilli. The finding of the latter is often necessary to establish a diagnosis. In all instances of pyuria, renal tuberculosis should be suspected. Catheterization of the ureters may show what organ is affected. The urine should then be centrifugated, and the sediment examined for bacilli, and, as Reynolds advises, a portion inoculated into guinea-pigs. The tuberculin test may be employed. The testicles and bladder should be carefully examined for primary tuberculosis.

Tuberculosis of the kidney presents symptoms like those of *pyelitis*, *renal calculus*, or a *new growth*. It is almost impossible to distinguish any one of the four until an interval has elapsed. In all cases the patient suffers from dull pain, sometimes with a bearing-down sensation. Hæmaturia occurs, and the patient is liable to attacks of renal colic. These symptoms may continue until a tumor can be made out. Even before this pain, which may extend all along the urinary tract, will be elicited on palpation. With the occurrence of the tumor the general symptoms of tuberculosis arise. Further diagnosis is based upon the results of the urinary examination.

Sarcoma and Carcinoma of the Kidney.

Either disease may be primary or secondary. Sarcoma may be congenital. The tumor may occur at any age, but is relatively common in young children. Twenty-five out of sixty-seven cases collected by William Roberts occurred in children under ten years of age. In older persons it is often preceded by calculus.

Symptoms. In some instances there are no symptoms during life. In others the disease may advance considerably before it presents any signs. If symptoms are complained of, they are usually limited to pain, the occurrence of hæmaturia, or the development of a tumor. The pain is dull and seated in the lumbar region. It may be neuralgic in character; and, indeed, there may be a true *sciatica* with paresis of the leg from pressure of the tumor. The *tumor* (Plate XLVII., Fig. 2) is firm; its surface is smooth or nodulated. It may be felt in the loins, and in front above the umbilicus, a few inches to the right or left of the median line; the descending colon lies in front of the tumor. The latter may grow with great rapidity and attain an enormous size, filling the abdominal cavity and giving rise to pressure-symptoms in surrounding organs. The growth

occurs more often anteriorly and downward toward the pubis because there is less resistance in these directions. As rapidly growing cancers are soft, the tumor frequently exhibits a certain degree of elasticity which may be mistaken for fluctuation. It is movable either by the hands or with respiration. On percussion the resistance is increased and the note is dull, except in front, where the colon, which has been pushed forward, gives a tympanitic note. If the colon should be flattened out between the tumor and the abdominal wall, it may be felt as a band stretching across the tumor, with dulness on percussion. In such a case inflation of the colon will be of great assistance in the diagnosis. Rare physical signs are pulsation and a blowing murmur. The *hæmaturia* may be constant or intermittent. The clots of blood may cause renal colic.

The *general symptoms* are those of carcinoma. A marked rapidity of the pulse has been noted in several cases. In girls a premature development of hair on the pubes and in the axillæ and pigmentation of the skin have been observed.

Hemorrhage is an early symptom, and in the absence of nephritis or cystitis should always suggest tumor. It may occur early and may be intermittent or persistent. In some instances it occurs but once; usually it is repeated. When excessive, the growth is never innocent. Pain is not of much diagnostic value, and may be absent until perinephritis occurs. Symptomatic *varicocele* may occur. The examination of the urine, save that it discloses the presence of blood, is negative. In this sense it is of value. Pus occurs if there is secondary infection or if calculi precede the growth. Rarely fragments of carcinoma are said to be detected. In order to determine which kidney is affected, the urine should be obtained separately from each organ.

The tumor must be distinguished from tumors of the lymphatic glands, of the liver, of the spleen, and of the ovary. It must not be confounded with psoas abscess or perinephric abscess, which causes a tumor in the lumbar region.

Cystic Kidneys.

1. Congenital. The kidney consists of a small mass of cysts filled with clear fluid. It may interfere with the birth of the child on account of its large size.

2. Acquired. The causes are trauma and obstruction of the ureter, the presence of which is determined by catheterization. The symptoms are those of a fluctuating renal tumor. The urine may be normal, or hæmaturia may be present.

Hydronephrosis.

Hydronephrosis may be congenital; it may be caused by obstruction of a ureter by a stone; the pressure of a tumor; twisting of the ureter, as in movable kidney; or represent an exudation.

Symptoms. In addition to the symptoms of the causal condition we have, upon the development of hydronephrosis, the presence of a *tumor* arising in the region of the kidney and extending toward the middle line.

Sometimes fluctuation can be detected ; often it can not. Variations in the size of the tumor may occur with changes in the amount of urine passed. Exploratory puncture and the finding of a fluid with elements of urine in it are valuable means of diagnosis ; but if the hydronephrosis is old, this fails, as the fluid loses its urinary character, and can not be distinguished from that of an ovarian cyst, for instance. When on one side, the urine may be normal ; when on both sides, it is diminished ; anuria and uræmia may occur. If pyelitis is present, pyuria is observed.

Pain may or may not be present. Gastric symptoms are very common. Either constipation or diarrhœa is seen. Hypertrophy of the left ventricle may occur, as in *chronic* nephritis.

Hydronephrosis consists in a dilatation of the kidney pelvis with urine, which is prevented from escaping by obstruction of the ureter, either by the pressure of a tumor or by disease of the bladder or ureter itself. In time the kidney atrophies from the pressure and a large cyst forms. The tumor has the physical characteristics of pyonephrosis, but the history is different, and if there is any discharge, it is free from pus. As in pyonephrosis, the tumor may become small after a copious discharge—in this case of urine ; or may even wholly disappear if the obstruction is removed. This sign is pathognomonic.

Intermittent hydronephrosis is associated with *movable kidney*, hence it is more frequent in women. It is characterized by the development of a renal tumor which disappears at varying intervals, pain, nausea, and vomiting ; during this period the urine is scanty. In a few hours or days there is an increase in the amount of urine and the tumor subsides.

Diagnosis. If absolute obstruction continues, the diagnosis must be made by the detection of a fluctuating renal tumor, the absence of fever and signs of suppuration, and by the result of exploratory puncture. The urine is usually free from pathological changes.

Hydronephrosis when very large may be confounded with ascites, but hydronephrosis is rarely bilateral, and the fluid does not change its level upon change of position of the patient, as is the case with ascites. The history of the two conditions is different.

An *ovarian cyst* can usually be traced into the pelvis ; it does not carry the colon in front of it, and hence is dull, even on superficial percussion, and it leaves the loins resonant.

Pyelitis. Pyonephrosis.

Pyelitis. The antecedent causal factors of pyelitis, which is rarely primary, are : severe infectious diseases (typhus, variola, diphtheria, pyæmia) ; toxic substances ingested (cantharides, etc.) ; chronic nephritis ; inflammation of the bladder or ureter ; strictures of the ureter or urethra ; hypertrophy of the prostate ; spinal palsies of the bladder ; calculus ; parasites ; and blood-clots ; infection being the active cause.

Symptoms. *Urine.* Pus in the urine with pelvic epithelium—although it is not safe to base a diagnosis on the presence of the latter ; casts of the canals opening into the pelvis, which are more characteristic ; epithelial casts and casts containing micro-organisms. The urine is often increased,

acid, and contains pus and albumin, rarely blood. *Pyuria* may be the only renal sign. In all forms of pyuria above the bladder Kelly withdraws the pus by catheterization and suction. He allows the catheters to remain for from ten minutes to four or five hours, in order to estimate the functional power of each kidney. Of course, the pus is studied microscopically and bacteriologically. *Pain* in the region of the kidney, often severe, is complained of, although it may be absent. When present, it is often of a tearing character. A *tumor* is often present, most prominent in the loin or in the abdomen. In the latter the mass can be felt two inches to either side of the umbilicus, usually above the transverse line.

Abscess of the Kidney. Pyelitis differs from abscess of the kidney. The latter may be the result of a local infection from the pelvis of the kidney or may be pyæmic. In abscess of the kidney there is some fulness in the loin of the affected side. The kidney is felt to be enlarged, and is tender and painful. A tumor may be detected anteriorly. The diagnosis is based on a study of the cause (acute nephritis, pyæmia, impacted calculus in the ureter, erysipelas) or the detection of blood and pus in the urine, which is scanty, and on the constitutional symptoms. The progress of the case is usually acute. If the abscess is *tuberculous*, tubercle bacilli can be detected in the purulent sediment of the urine, and there will be other foci of tuberculosis with a corresponding clinical history.

Pyonephrosis. When the pus is confined by an occluded ureter, the pelvis is over-distended. In pyonephrosis the *tumor* is tense, smooth, and globular. Fluctuation may be detected. Tenderness is usually absent; the course is slow and does not affect the general health so much as abscess. The pus may be discharged copiously from time to time, and the tumor be therefore diminished in size. The urine may be occasionally almost clear. *Fever* is irregular, remitting, or septic. The fever and pyuria may be the only symptoms. If the *bladder* is healthy, vesical symptoms fail to aid in diagnosis. Pyonephrosis arises secondarily to pyelitis, and often after the latter has lasted some time.

Perinephric Abscess.

This may occur as a primary disease in apparently healthy individuals, or follow one of the infectious diseases.

Perinephritis arises usually from extension of inflammation and supuration from the kidney, but may be the result of strain, exposure to cold, or injury. Perinephritis may also be pyæmic, and occur after infectious fevers and in actinomycosis.

Symptoms. The **secondary forms** have symptoms of the primary disease, and, later, swelling and pain in the renal region.

Primary Form. Chills and fever, pain, difficulty in defecation. The general condition suffers. Finally, in all cases, a swelling, hard at first, develops in the lumbar region; then œdema of the skin follows, and fluctuation is detected. The abscess may descend and point above Poupart's ligament. It may press upward and cause dyspnoea. Great tenderness and pain in the region of the swelling may arise, and the pain

may radiate to the leg. Irregular septic fever and chills appear. The *urine* is not generally changed unless there is some communication with the pelvis or ureter. The patient lies on his back, turned toward the affected side. The knee and hip of this side are flexed, and the thigh is rotated outward. The affection may simulate *coxitis* and *appendicitis*.

The *swelling* of a perinephric abscess appears in the lumbar region of the side affected. It is rounded in form and doughy. (Da Costa.) Like other kidney tumors, it is not affected by respiration. The usual signs of confined suppuration exist, and pulmonary or pleural complications may occur. As the abscess progresses, the local signs of suppuration become more marked, the skin reddens, and pus may be discharged externally.

The most marked subjective symptom is *pain*, which may amount to agony, and is paroxysmal; soreness from restricted motion of the psoas muscle is apt to be complained of.

A *tumor* was present in the loins in 65 out of 71 cases analyzed by Fenwick, but did not generally manifest itself until the inflammation had made considerable progress. There is dulness on percussion even in the early stage, and later, fluctuation. The general symptoms are vomiting, constipation, fever, and sometimes rigors. The disease is more common in males than in females (61 males to 39 females in Fenwick's cases).

Hydatid Disease.

A *hydatid cyst* of the kidney presents the usual physical signs of such cysts. A fremitus may be detected, or small cysts may be found in the urine.

The disease is comparatively rare. Usually there are no symptoms until a tumor is felt. Then pain gradually develops. The cyst may open into the pelvis of the kidney, and cysts or scolices be discharged, with *colic*.

Pyelitis and cystitis may also develop.

Echinococcus cysts may become inflamed and lead to general pyæmia. Puncture of the discovered tumor, which should be performed in most cases, is otherwise the only means of diagnosis. It must be differentiated from hydronephrosis and ovarian tumors.

Nephrolithiasis (Renal Calculus).

Renal calculi vary in size from "sand" through "gravel" to "stones." The last may be from the size of a cherry to one large enough to fill the pelvis of the kidney. They consist usually of uric acid, and are hard, brownish-red or blackish, and crystalline; the larger ones are arranged in distinct layers. More rarely we have calculi of calcium oxalate, extremely hard and nodular. Some stones have alternate layers of the two salts; others consist of phosphates, but usually the centre is of uric acid or calcium oxalate, the phosphates having been deposited after the urine became alkaline. Very rare forms are composed of cystin, xanthin, indigo, etc.

Frequency. A consideration of the frequency of the affection and some ætiological data aid in the diagnosis. It is not a common affection. I have had 29 cases in private practice and 11 in hospital practice (1898). Thirteen cases only have been treated in the Presbyterian Hospital in twenty-five years, during which time over 8000 cases of all kinds were treated.¹ It is a disease of the middle and upper classes. This is particularly true of uric acid calculus. It is not, in my experience, a disease of the old or the very young. The youngest subject was twenty-five years of age; the oldest, sixty-nine. Twelve of my private patients were of the female sex, seventeen of the male sex. There does not seem to be much difference between the two sexes in regard to frequency. Most authorities, however, hold to the preponderance in women, the ratio being as 3 is to 1. Sedentary occupation and an indoor life are predisposing factors.

Symptoms. With the exception that some present no symptoms whatever, the cases may be divided into three groups:

a. Calculi may remain in the pelvis of the kidney, and not cause any renal symptoms. They may cause gastric disturbance or catarrh of the bladder or renal pelvis. There may be occasional pain in the lumbar region, the cause of which is unsuspected.

b. The calculus may excite pain, hæmaturia, and frequent micturition.

c. The stones may attempt to pass from the pelvis of the kidney into the ureter, causing renal colic, the symptoms of which have been described above. In the intervals between the attacks of colic the patient may be free from symptoms.

The symptoms ascribed to the presence of a calculus in the pelvis of the kidney are *pain, intermittent hæmaturia, pyuria, pyelitis, renal intermitting fever, acute orchitis, frequent micturition, and renal colic.*

Pain. Pain of the affected organ is the most constant symptom, and is increased by movement, by jolting, and by pressure. Indeed, pain induced by pressure is of as great significance as spontaneous pain. It frequently is persistent, and even continues in any position assumed by the patient. The pain is seated not only in the regions just indicated, but extends also along the ureter, from the loin to the front of the abdomen. It may persist for some time, at a point on either side of the umbilicus above or below it, or at a point on the surface of the abdomen opposite the brim of the pelvis. Thence the pain radiates into the bladder, either above the pubis (the hypogastric region) or into the testicle, or down the inside of the thigh. It may be in the loin and at the end of the penis at the same time, or cause lancinating irritation along the whole urinary tract. In rare cases the pain is in the kidney of the healthy side. The pain of renal colic is always associated with frequency of micturition, with or without pain during the passage of the urine. Unlike that of renal calculus, it comes and goes, and is more commonly intermittent and paroxysmal. Very frequently, however, it is constant and localized either in the region over the kidney or anteriorly in the region mentioned. In my experience it comes on during the day, particularly the after-part of the day, and not, as Jacobson would have us believe, at night. That it may occur spontaneously is not so much a

¹ "Renal Calculus," J. H. Musser, Philadelphia Medical Journal.

peculiarity of renal calculus as that it can be excited by pressure or movement.

Pain is of greater diagnostic significance in *renal calculus* than in any other renal affection. Every attribute that has been applied to pain belongs to the pain of renal calculus. Its very vagaries render its presence one of the most valuable signs. Often, however, it behaves like the flitting nerve-aches of hysteria, and we must see to it that this counterfeit is not passed upon us. Urinary phenomena do not serve for the distinction; other neurotic manifestations or the stigmata of hysteria aid in the diagnosis. The pain may be aggravated by the function of menstruation and even bear a close relationship to it.

Hæmaturia. Hemorrhage from the kidney is the classical symptom of stone. It is the most constant and positive symptom of renal calculus. Prior to the use of the centrifugal machine, blood, when in small amounts, no doubt escaped the eye of the observer, partly because it was destroyed as the urine advanced in decomposition during the period it was set aside for sedimentation and partly because the small number of the corpuscles rendered them difficult to find. Excluding all causes outside of the kidney—*i. e.*, of vesical and ureteral origin—renal hæmaturia may be due to congestion and inflammation, to infarct, to new growths, to tuberculosis, to renal calculus, or to parasites. The fevers and infections, and scurvy, purpura, leukæmia, and hæmophilia are responsible for a number of cases.

In six years 2923 samples of the urine of 1997 persons were critically examined in my laboratory. Blood was present in 364 cases, detected by microscopical examination alone. The hæmaturia resulted from congestions or hyperæmias (pregnancy, goitre, heart disease, the fevers, infections, and jaundice) in 56 cases. In 42 cases the hæmaturia occurred in the course of acute and chronic Bright's disease, and in 19 more in arteriocapillary fibrosis, either of renal or of cardiac origin. Gastric disorders, rheumatism in many forms, gout, neurasthenia, and anæmia account for 81 of the cases, conditions always associated with the copious discharge of urinary salts, which are irritating. Vesical disease accounts for 17 cases, renal calculus for 28, and in 20 the diagnosis was not noted at the time and is forgotten. All the cases of renal calculus had hæmaturia. It is not an intermittent phenomenon alone, but one that is constantly present.

It is necessary to eliminate all sources of urethral, vesical, and ureteral hemorrhage before coming to a conclusion that the hemorrhage is of renal origin. *Cystoscopy* must be resorted to, of course, and possibly *ureteral catheterization*, which demands expert skill. If the hemorrhage is free, the time of its passage in the act of urination must be determined. The reaction of the urine must be borne in mind. It is true that catheterization alone may suffice to determine which kidney the hemorrhage comes from. *Blood-cylinders* are rarely if ever present in renal calculus. They denote hemorrhage from the renal substance.

In a person of middle life that may be supposed to have, by virtue of heredity, occupation, and habits, uric acid or oxalic acid tendencies, and in whom no cause for the hemorrhage can be found in the urethra, blad-

der, or ureter, the chances are that it is of pelvic origin, due to the irritation of gravel or of urine heavily loaded with salts.

Klemperer¹ has recently called attention to hæmaturia from healthy kidneys as the result of over-exertion, in one case from horseback-riding, in another from bicycling. He also reports 4 cases of hæmophilia and a group due to an angioneurosis. Hyaline casts were not present, although blood-cylindroids were. General symptoms of neurasthenia supported the diagnosis in the angioneurotic cases.

Pyuria. Pus in the urine is looked upon by all authorities as almost essential to the diagnosis of renal calculus, but in my experience this product of inflammation is usually absent. Of the 28 cases which I have examined in 15 there was no pus; in 6 a few cells or a very small quantity was found (4 uterine, cause obvious); in 1 it was noted as considerable (old gonorrhœa and syphilis, 4 examinations); in 1 a small quantity (male, cause assignable); in 1 it was small in amount—twice only in some 50 examinations; in 1 it was abundant and due to genito-urinary infection as well as to pyelitis. Pyuria is not present unless an accidental infection has taken place from the lower tract.

Albumin. In 21 patients albumin was found. It was in large excess in 3, due to coexisting Bright's disease. As a trace it is of frequent occurrence and does not imply a coexisting nephritis.

Urinary Casts. Casts are present in the urine in nearly all cases of renal calculus. The specimen must be centrifugated. They are hyaline—not abundant—long and narrow. Their persistence without other kinds, with or without albumin, is diagnostic of renal irritation, and with other signs points unfailingly to calculus.

Specific gravity of the urine is an aid in the diagnosis. Its persistence above the normal is both a comfort and a sign. It enables one to exclude renal cirrhosis and eliminates hysteria or a renal neurosis.

Frequent micturition is not in my experience an indication of stone in the kidney, save when attempts are made to pass the stone (colic), although it is spoken of as a symptom of value by most authorities.

Paroxysmal renal fever, allied to hepatic fever in its expression, rarely occurs, but when present, may be due to calculus. It may also be due to absorption of retained products if the kidney is floating and becomes twisted. It may be due to pyelitis.

Duration of symptoms and family history are also valuable data.

Diagnosis. Middle life is a predisposing factor, and persistent hæmaturia is symptomatic, but pyuria rarely so, while albuminuria and hyaline casts in urine of high specific gravity are prominent elements of the symptom-complex upon which a diagnosis is based.

The diagnosis can be established by the symptom-complex of *pain*, local *tenderness*, persistent *hæmaturia*, *albuminuria*, and *casts* (the cardiac, vascular, or renal origin of which is excluded), by the phenomena of *renal colic*, and by the *passage of fragments* of stone.

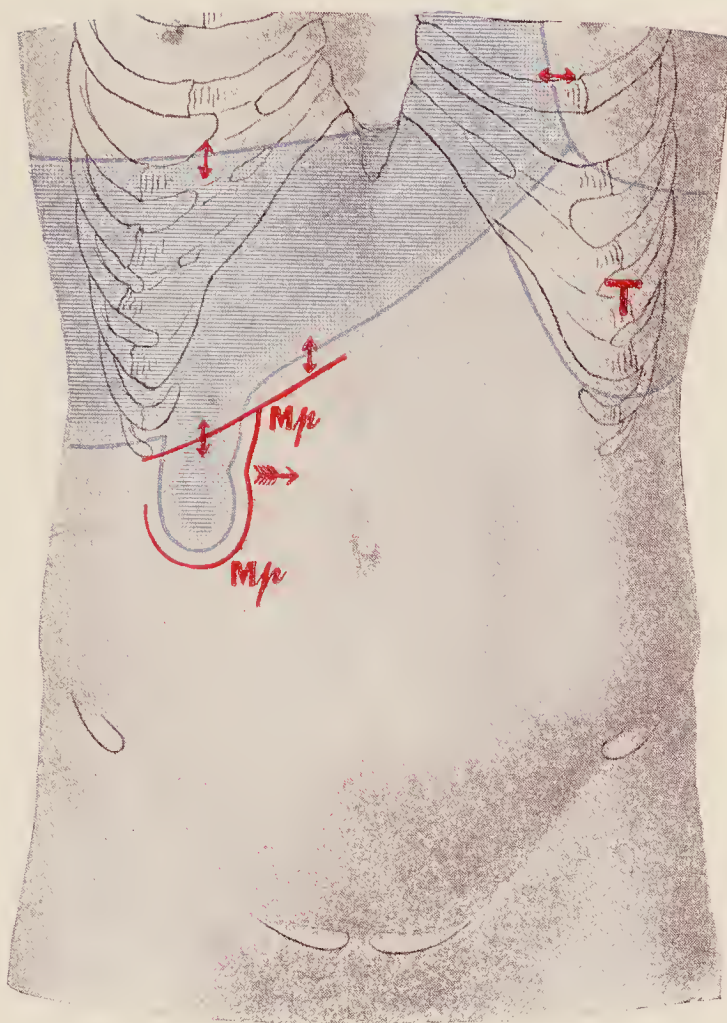
If the hemorrhage persists after prolonged rest, it is more likely of cancerous or tuberculous origin.

The *differential diagnosis* must be made from *appendicitis*, *movable* and

¹ Deutsche med. Wochenschrift, March 4, 1897.

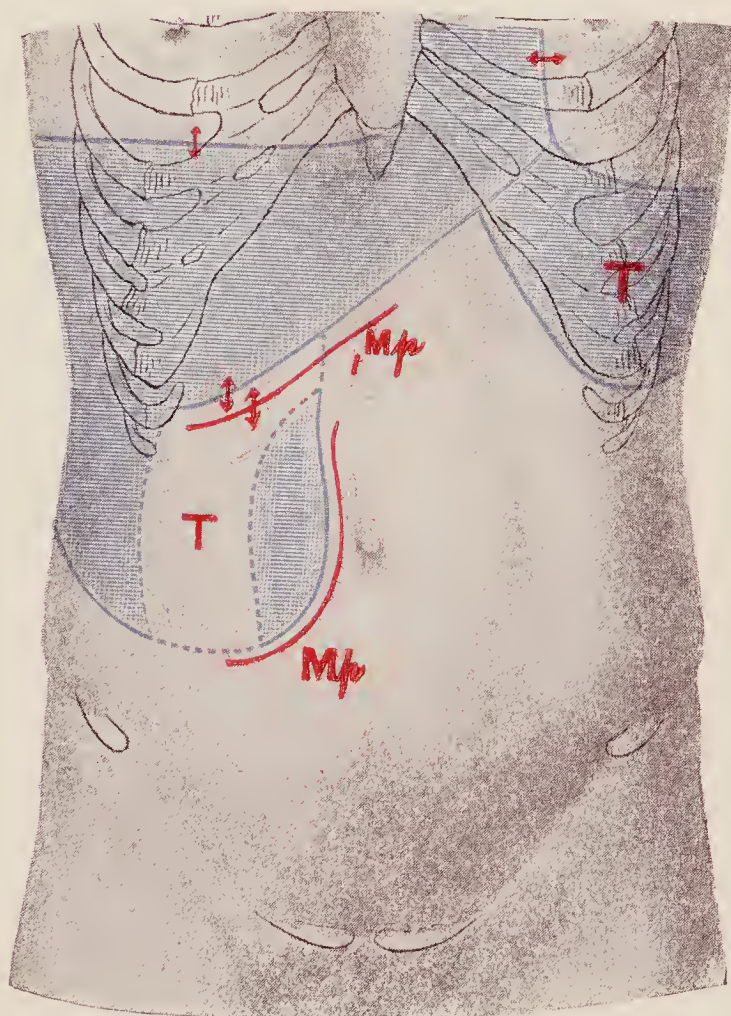
PLATE XLVII.

FIG. 1.



Movable Kidney.

FIG. 2.



Sarcoma of the Right Kidney.

twisted kidney, biliary colic, and other affections that simulate these conditions. Catheterization and exploration through the ureter are required in many cases. Hollander believes that in a large number of cases a diagnosis can be made without the aid of catheterization, and fears the danger of infection from below.

Kelly performs ureteral catheterization very skilfully and aspirates the ureters, thereby bringing down fragments of calculi. He also explores the ureters with hard-rubber bougies tipped with wax. He can determine the presence of calculi by the markings on the tip of the bougie.

Movable Kidney.

Movable kidney is usually seen in women after the age of forty years, who have done manual work or have borne many children.

It is frequently preceded by a history of injury, unusual lifting or strain, followed by tearing or dragging sensations in the abdomen. Pain may continue for several weeks after the injury and then subside, and the accident be forgotten, or subjective sensations may continue.

In other instances the movable kidney is part of a general visceral displacement seen in men, though more frequently in women. Gastropsis and gastro-enteroptosis can usually be made out in such cases.

Symptoms. The symptoms are due to the local dragging or pulling of the kidney on its bloodvessels and nerves, or to reflex symptoms, or to pressure upon adjacent organs.

The *pain* of movable kidney is usually referred to the right or left of the median line; sometimes to the hypogastrium. It may be constant, dull, and aching in character, aggravated at intervals; or it may be paroxysmal. The paroxysms continue for three or four days, during which time other subjective symptoms arise. The attacks are known as *Dietl's crises*. Nausea may attend the paroxysms, or be more or less constant. Sometimes vomiting takes place. Swelling and tenderness of the kidney are found. The pain, inducing shock, vomiting, and local tenderness, may simulate peritonitis. Instead of pain, a dragging sensation is experienced. The patient may be aware of the presence of a tumor or lump in the abdomen as well as of its movability.

The *reflex* symptoms are chiefly referable to the nervous system. Emotional disturbance is observed when the organ is displaced. Hysteria may be present. Palpitation of the heart is a common reflex symptom. There are often depression of spirits and hypochondriasis. *Jaundice* may occur from pressure, and the intestine may be occluded from the same causes.

Urinary Symptoms. At the time of a crisis the urine is scanty and hydronephrosis may develop. As the kidney slips back into its bed, the twisting of the ureter is relieved, and copious discharges of urine take place. Polyuria and perhaps frequent micturition are not uncommon.

Objective Symptoms. (Plate XLVII., Fig. 1.) The abdominal walls are usually relaxed, and may or may not contain a large amount of fat. *Movable kidney* is best detected by palpation. The patient should stand with the body bent forward and the hands resting on a chair, as described

elsewhere. The organ is recognized by its rounded borders, its bean shape, its movability, the detection of the hilus and perhaps of the pulsation of vessels in it, and by the fact that it can be replaced. Palpation causes a sickening feeling analogous to that experienced when a testicle is compressed, but less in degree. Percussion will demonstrate that a body, supposed from palpation to be the kidney, is a solid organ. The tumor can be found to the right or left of the median line, freely movable and changing its position with that of the patient. If the tumor is situated on the right side, it may be in close proximity to the liver, or be felt opposite the umbilicus, or often in the iliac region. When near the liver, by careful palpation the fingers can be introduced between the border of the liver and the mass. Usually it does not move with respiration, but sometimes it is found to do so. On the left side it may be as high up as the margin of the ribs. It is generally felt in the midclavicular line, a little above the level of the umbilicus.

In a case recently under the writer's care the woman, aged fifty-five years, experienced pain in the abdomen about once a month, to the right of and above the umbilicus. At times nausea and vomiting accompanied the attacks; at other times marked depression or hysteria. Anuria always occurred and continued for a variable time—not longer than five days. With one of the paroxysms a tumor was found in the region of the gall-bladder, movable with respiration, but distinctly defined from the liver by placing the fingers between the lobe and the kidney. It moved with each change of position of the patient, and at first the hilus could be distinctly felt. As the pain continued, the anuria persisted and a marked change in the tumor was observable; it gradually increased in size, and a portion of it fluctuated; it was round and partook of the character of a cyst. The fluctuation was detected by placing the hand on the tumor in front and pressing firmly toward the other hand placed in the loin above the pelvis. After several days a copious discharge of urine took place and the swelling subsided.

Diagnosis. Movable kidney may be confounded with tumor of the *gall-bladder*, tumor of the *pylorus*, and with tumors in the *pelvis*. It is not likely to be confounded with an omental tumor, carcinoma, or tuberculosis, because the phenomena of these processes are not present and ascites does not occur, nor is there rise of temperature, as in many cases of tuberculosis. As pointed out by Henry Morris, tumor of the gall-bladder and movable kidney are frequently of conjoint occurrence. Movable kidney is distinguished by the absence of previous history or of symptoms or signs indicating disease of the gall-ducts. If jaundice is present, it is not so intense as in tumors of the gall-bladder. While the gall-bladder is movable, it is less distinctly so than movable kidney. The gall-bladder moves in an arc of a circle, the centre of which is at the edge of the right lobe of the liver. It can be pushed further to the left than to the right, but never downward like a movable kidney. Moreover, the gall-bladder is always palpable; the movable kidney can not always be felt. The gall-bladder, if it contain calculi, is very hard compared to the kidney. Anuria does not occur. The kidney tends to spring back to its place in the loin; the gall-bladder to the anterior part of

the abdomen. Even if the gall-bladder is enlarged, the kidney may be felt by bimanual palpation; while the opposite does not obtain. In cancer of the pylorus the emaciation and anæmia are more pronounced than in movable kidney. The vomiting usually present in that affection and the physical signs of dilated stomach can be made out. Tumors of the pelvic organs are determined by examination according to the usual methods.

CHAPTER XII.

DISEASES OF THE MUSCLES.

Myalgia.

MYALGIA is an inflammation of the muscles produced by a "cold," by trauma, or by rheumatism. There is pain on movement and spontaneous pain in the muscle ; it is tender on pressure, and may be the seat of spasm.

Muscular Rheumatism.

In this variety of rheumatism there is pain in the affected muscles, which often comes on suddenly in the night or is first noticed when the patient attempts to rise in the morning. The pain when the patient is at rest may be inconsiderable, rarely amounting to more than a dull, aching, sore feeling ; on attempting to move, bend, twist, or straighten himself, however, the patient catches himself suddenly on account of the agonizing, tearing, or burning pain. When the muscles are relaxed, the patient is fairly comfortable. Sudden movement is the most painful. The affected muscles are tender to the touch and to sharp blows. Muscular rheumatism may be acute or chronic. In the latter the symptoms are very much like those of chronic articular rheumatism, except that the muscles and not the joints are affected. There is the same proneness to recur in unfavorable weather and in cold, damp seasons.

The disease receives different names according to the muscle affected. The most common subvarieties are : *lumbago*, in which the muscles of the small of the back are affected ; *pleurodynia*, in which the intercostal muscles suffer ; and *torticollis*, in which the sternomastoid and trapezius are painfully contracted.

In *lumbago* the patient holds himself rigid and is unwilling to rotate the trunk upon the vertebræ. Often the most comfortable position is that in which he sits and bends slightly forward over another chair. Motion is painful, but pressure is not. Fever is absent. There is a history of repeated attacks or of exposure, such as lying upon damp ground. Lumbago needs to be distinguished from disease of the spinal membranes, from disease of the vertebræ, aneurism, abdominal abscess, and diseases of the uterus and ovaries. The diagnosis of rheumatism is arrived at by exclusion.

In *pleurodynia* there is usually tenderness upon pressure as well as upon motion and deep inspiration. The pain is of the same sore, burning character, aggravated by coughing and sneezing. The patient breathes as little as possible, and often bends over toward the affected side to lessen the motion. Pleurodynia is distinguished from pleurisy by the absence

of fever, cough, and, above all, of friction-sounds. In intercostal neuralgia there are painful points upon pressure, whereas in pleurodynia firm pressure is grateful, though tapping is painful.

In *torticollis* the head is drawn to one side and fixed in that position. The sternomastoid especially is rigid and tender on pinching. In spinal affections the head is retracted, and there are antecedent symptoms, as headache and darting pains with fever.

Thomsen's Disease (Myotonia Congenita).

This is an hereditary disease, and may occur in several generations of a family. Tonic cramps take place in the muscles when voluntary movements are attempted. The disease begins in childhood, rarely after puberty. The muscles become rigid and fixed when put in action. The lack of voluntary control of the muscles is shown by the slow contraction and relaxation when voluntary efforts are made. The rigidity may wear off and the limb can then be used. It is particularly noticeable when walking is attempted. As the leg is advanced slowly it may remain stiff for a second or two, but after it becomes limber the patient can walk for hours. If he stops walking, the same difficulty is experienced when he starts again. Both arms and legs are affected. Patients are usually well nourished, however. There are no atrophies. The muscles are irritable, so that mechanical stimulus or pressure causes tonic contraction. Movement and cold aggravate it. Sensation and the reflexes are not affected, and there is no evidence of disease of the cerebrospinal system, save the occurrence of hypochondriasis in some cases. The myotonic reaction described by Erb is induced. (See electrical diagnosis—Diseases of the Nerves.)

Paramyoclonus Multiplex.

In this affection there is clonic contraction of the muscles. It is usually confined to the extremities, and occurs in paroxysms. It may have been caused by sudden twitching or violent motion. The clonic spasms at first do not interfere with the patient's occupation, but gradually they increase. Both legs are affected, and the number of contractions varies from 50 to 150 a minute. The contractions may be rhythmical. In severe cases the muscles of the back and abdomen contract violently. Tremor of the muscles may be present in the intervals. (For *paralysis, spasm, tremor, contraction*, etc., see Diseases of the Nervous System.)

Myositis. Inflammation of the Muscles.¹

In inflammation of the muscles there are pain, swelling, and loss of power. In universal myositis the inflammation begins in the lower extremities and gradually involves other muscles of the body. They are swollen, hard, and painful on pressure. Atrophy supervenes in groups of muscles. The muscles may become more or less rigid. Local œdema of the skin over the muscles occurs. The progress is gradual, and death ensues when the respiratory muscles are involved.

¹ See also Trichinosis.

The three cardinal symptoms that attend the disease as described by Löwenfeld are : (1) swelling of the extremities due to subcutaneous œdema and swelling of the muscle, causing functional disturbance ; (2) extension to the muscles of respiration and deglutition ; (3) a more or less extensive eruption. This last is erythematous, its distribution is usually general but irregular, and may be followed by pigmentation. The disease must not be confounded with *trichinosis*. In the latter, examination of a small portion of muscle reveals the trichinæ.

Progressive ossification of the muscles is rare. The muscle tissues undergo gradual ossification, either in localized spots or in widespread areas. Inflammation of the muscle precedes the ossification. As the inflammatory swelling subsides, the muscles become hard and are gradually converted into bony tissue. The disease lasts many years.

CHAPTER XIII.

DISEASES OF THE NERVOUS SYSTEM.

THE semeiological classification of nervous diseases presents many difficulties. Many forms that are closely analogous in their symptoms are widely different in their pathology or ætiology, and many diseases present such variations in their symptom-complex that at one period they could properly be placed in one group and at another period elsewhere. In general it may be said, however, that the diseases of the peripheral motor neurons differ so widely from those of the central motor neurons that they can be classified as two separate groups, and in a third group would come the diseases of the sensory neurons. Combinations of these three groups, producing on their part rather clearly marked complexes of symptoms, may then be described, and finally the general and local diseases of the brain and cord. An entirely separate group, characterized by peculiar symptoms, comprises the so-called functional nervous diseases, or the neuroses.

DISEASES OF THE PERIPHERAL MOTOR NEURONS AND THE MUSCLES.

DISEASES CHARACTERIZED BY PURE MOTOR DISTURBANCE.

Progressive Muscular Atrophy.

Two forms are recognized—the *scapulohumeral type of Erb* and the *facio-scapulo-humeral type of Déjérine-Landouzy*. The scapulohumeral type of the disease commences about the age of puberty in the majority of cases. It is frequently distinctly hereditary or familiar, cases having occurred either among the ancestors or in other members in the family in the same generation. The onset is very gradual; usually the muscles of the shoulder are first affected, especially the pectorals and the latissimus dorsi. Next the adjacent muscles are involved, followed by the muscles of the arms, thighs, and finally those of the calf. There is gradual loss of power corresponding to the atrophy of the muscles, but reactions of degeneration do not occur. As a result of the wasting, peculiar alterations appear in the configuration of the body—that is, the shoulder-blades become prominent, lordosis develops, and, as a result of the weakness of the glutei, it may be necessary for the patient to rise, as in the pseudohypertrophic form, by climbing up his legs. The gait, as a result of the atrophy of the quadriceps, is waddling in character. Sensory disturbances are absent. The reflexes are not altered in the early stages, but as the muscles gradually atrophy and become weaker, the ten-

don-reflexes undergo a corresponding diminution. The electrical reactions remain normal until late in the course of the disease, and even then show only a diminution of response proportional to the degree of muscular atrophy. In doubtful cases, or for the purpose of confirming a probable diagnosis, a small portion of a muscle may be excised and examined for the characteristic histological changes. This is most easily accomplished with the muscle-harpoon.

The *facio-scapulo-humeral type (infantile form)* commences earlier in life—about the third or fourth year. It is also hereditary, and the symptoms are essentially the same, excepting that the earliest sign of atrophy appears in the muscles of the mouth and the eyelids. As a result, the patient acquires a peculiar and somewhat characteristic expression. The eyes are partially open even during sleep, the lips remain apart, and saliva dribbles from them. Ultimately, the other muscles of the body are involved, and the disease assumes the characteristics of the preceding form.

Pseudohypertrophic Muscular Dystrophy.

This disease commences in early life—from the third to the sixth year. Ordinarily, the muscles of the calves are first involved. These become greatly enlarged, hard, and there is great loss of power. Other muscles of the legs are next involved; then those of the back, and perhaps the arms. Not all the muscles that undergo atrophy show a preliminary hypertrophy. The electrical reactions remain normal, and the loss of power is due merely to the atrophy of the true muscle-substance. The gait is waddling, and the patient is unable to arise from the ground except by getting upon the hands and knees and then gradually climbing up his legs. There is usually lordosis or scoliosis, and occasionally contractures occur, leading to formation of club-foot. Sensation remains unimpaired throughout the disease. The reflexes show diminution corresponding to the loss of muscular substance. The intelligence of the patient in this, as in the two preceding forms, remains intact. The course is slowly progressive.

DISEASES CHARACTERIZED BY MOTOR DISTURBANCE, WITH DEGENERATIVE CHANGES IN THE MUSCLES.

Progressive Neural Muscular Atrophy. (Charcot-Marie Hoffmann Type. Peroneal Type of Gowers.)

This is a disease of early life, the first symptoms appearing just before puberty. The muscles first affected are those of the feet and hands; usually in the former, the peronei, the extensors of the toes, and the small muscles of the foot; in the latter, the interossei and the muscles of the thenar and the hypothenar eminences. The affected muscles show distinct fibrillary twitchings and usually the characteristic reactions of degeneration to the electrical current. These reactions of degeneration are also present in the nerves. There is usually a coarse, irregular tremor, and the atrophy of some of the muscles with contractures of others gives

rise to various deformities, such as the *ape-hand*, the *main en griffe*, or, if the foot is first affected, to *foot-drop*. Later the foot assumes the position of equinovalgus or equinovarus. In this disease there is sometimes involvement of the sensory fibres, and the patients may complain of slight paræsthesia or even of pain, but the nerve-trunks are not sensitive. Hypæsthesia is also occasionally present. The process usually is restricted to the limbs, the muscles of the trunk and face escaping; bulbar symptoms are almost unknown, and the functions of the bladder and rectum are not disturbed. In a form of this disease described by Déjérine under the title of *Infantile Hypertrophic and Progressive Interstitial Neuritis* there are, in addition to the above changes, the symptoms of locomotor ataxia—that is, Romberg's symptom, lancinating pains, ataxic gait, and even disturbance of the pupillary reflexes. The nerve-trunks become enlarged and can be felt beneath the skin.

Progressive Spinal Muscular Atrophy. (Chronic Progressive Anterior Poliomyelitis. Type of Duchenne-Aran.)

The idiopathic form of this disease usually commences about middle life. The hereditary form appears, as a rule, somewhat earlier. The course varies somewhat, but in general is as follows: The first changes are observed in the muscles of the hand, particularly in those of the thenar eminences, giving rise to the formation of the *ape-hand*. The interossei and lumbrical muscles are next involved, and the interosseous spaces become deeper, the fingers become gradually weakened, and ultimately are fixed in a semiflexed condition—incomplete *main en griffe*. The muscles show fibrillary twitching and give the reactions of degeneration to the electrical current. The process affects first one hand, usually the right, and then the other. As the disease progresses it next involves the muscles of the shoulder, especially the deltoids, and later the muscles of the upper arm, and then those of the forearm. Finally, the muscles of the back, and even those of the lower extremities, become involved. Sensory disturbances are rarely present. Occasionally the patients complain of slight pains and paræsthesiæ. The reflexes are lost early. The course of the disease is exceedingly slow, and for a long time a considerable amount of compensation for the muscular disability is acquired. The emaciation is extreme, but total paralysis occurs only very late in the disease. Death ultimately results from the involvement of the intercostal muscles and the diaphragm, giving rise to respiratory failure. It is usually due to a terminal pneumonia. In addition to the typical course described, the disease may assume many other forms, and sometimes for long periods appears to be arrested.

Acute Anterior Poliomyelitis. (Infantile Spinal Paralysis.)

This ordinarily occurs in early life, between the first and twelfth years, but may appear considerably later. Frequently it is epidemic, and many cases may be observed in a limited district. It is really an infectious disease, commencing with chills, fever, headache, pains in the limbs and

back, and occasionally convulsions, and characterized by the rapid appearance of flaccid paralysis in one or more limbs. The onset is usually sudden, and the paralysis may occur before the development of the general symptoms. The legs are more frequently involved than the arms; the muscles are usually affected in functionally similar groups, such, for example, as the flexors of the upper arm. The paralyzes are more extensive at first than later, many of the muscles recovering after subsidence of the acute symptoms. The affected muscles are flaccid, atrophy rapidly, and, during the second week, begin to show the reactions of degeneration; when the atrophy becomes complete, all electrical response ceases. Secondary contractures usually appear in one or two months, and may produce deformities, particularly the various forms of club-foot, scoliosis or lordosis, and contractures of the hand. The tendon- and skin-reflexes are lost. Sensation is not disturbed. Circulation in the paralyzed limb is usually poor. The disease almost invariably occurs in children, and subsequently the affected extremity does not grow so rapidly as the other. Occasionally adults are attacked.

Chronic Anterior Poliomyelitis.

Chronic anterior poliomyelitis is characterized by the slow development of paralysis in one or more groups of muscles of the extremities of the body. The flexors are more frequently involved than the extensors. The muscles show fibrillary twitchings and give the reactions of degeneration, and the paralysis is usually flaccid. The process is usually self-limited, but bulbar symptoms may appear and cause death. The disease closely resembles progressive spinal muscular atrophy.

Muscular atrophies secondary to joint-lesions also occur. These are purely functional in character, and ordinarily there are neither reactions of degeneration nor fibrillary twitchings. Sometimes, however, both phenomena are present.

Periodic Paralysis. (Goldflam's Disease.)

This is a disease characterized by the occurrence from time to time of paralysis of all four extremities. The paralysis is usually flaccid in type, occurs without pain, and is associated with extraordinary increase in the electrical resistance of the skin. The disease usually occurs in several members of the same family, the paroxysms lasting three or four days.

DISEASES CHARACTERIZED BY DISTURBANCE OF MOTION OCCURRING WITHOUT REFERENCE TO ANY DEFINITE PORTION OF THE CENTRAL NERVOUS SYSTEM.

Chorea.

Sydenham's chorea is a disease of early childhood. The attacks may recur for a number of years. They are nearly always more severe in winter than at any other season. Girls are affected slightly more frequently than boys. It is characterized by irregular twitching movements

affecting various groups of muscles in the body that are usually functionally associated, so that the movements appear to be the result of voluntary innervation. These movements may be generally distributed, or more pronounced on one side than on the other, or may even occur in only one part of the body. They may involve the muscles of the face, the arm, the leg, or the muscles of the trunk, particularly the diaphragm, giving rise to an irregular, jerking inspiration. They may vary in severity from slight, almost imperceptible contractions to severe, general convulsive movements in which the violence is so great that bruises or even fractures may occur. They cease during natural or artificial sleep. As a rule, the affected limbs are slightly weaker, and in some cases this paralysis is very pronounced (*paralytic chorea*). The mind is usually clear, but there may be loss of memory or irritability of temper. In a few cases with violent movements there are pronounced insomnia and violent delirium (*chorea insaniens*). Speech may be affected either as a result of choreic movements of the lips or on account of psychic disturbance. The reflexes, sensation, and electrical reactions of the muscles are normal. Chorea is often associated with articular rheumatism and endocarditis, and is therefore probably an infectious disease. Occasionally in the violent form fever occurs.

Huntingdon's Chorea.

Huntingdon's chorea is characterized by the development, between the ages of twenty and forty, of choreiform movements of moderate degree, associated with gradually progressive dementia. The disease is strictly hereditary, occurring only in the offspring of those who have suffered from it; in some families it has been traced through five generations. The twitchings resemble those of chorea, but are rarely violent, and are often associated with a slight rigidity. The earliest mental symptom is usually loss of memory; later, there may be delusions of grandeur or melancholia. Usually life is prolonged to an advanced age, the mental symptoms gradually passing into the type of severe senile dementia. A curious feature is the tendency of the patient to avoid society.

Chorea Electrica.

There are several varieties of this condition—one occurring in children, characterized by lightning-like contractions of groups of muscles, sometimes those of the trunk or those of the extremities; another, *Dubini's disease*, which appears to be an infectious process, commences with violent pains in the head, neck, and back, slight fever, and general convulsions. Muscular contractions occur, usually involving all the muscles of the body. These attacks are characterized by their frequent recurrence and brief duration. Death is the usual termination.

Paramyoclonus Multiplex.

This is a disease, possibly hysterical in nature, characterized by lightning-like contractions in groups of muscles, which do not, however, produce movements that in any way resemble co-ordinated actions. Often

the patient from time to time emits a peculiar sound resembling a grunt, probably the result of diaphragmatic involvement. The electrical reactions are normal, and the reflexes are sometimes slightly increased.

Habit Spasm.

Habit spasm is characterized by the repetition of some peculiar, unnecessary movement, such as shrugging the shoulders, winking the eye, rubbing the elbow against the side, etc. Emotional disturbances or the presence of bystanders always increase the symptoms.

Saltatoric Spasm. (Jumpers' Disease. Latah.)

Saltatoric spasm is a hysterical manifestation in which the patient, whenever he or she attempts to stand, is compelled to rise on the toes or even to spring from the ground. Often after such movement the patient falls. The spasm disappears if the patient lies down, but may be produced by pressure upon the soles of the feet. It often occurs as an epidemic manifestation, affecting whole communities at once.

General Tic. (Maladie de Gilles de la Tourette. Maladie des tics convulsifs.)

General tic is a psychical condition characterized by curious movements of the limbs, grimaces, and the utterance of words that have no relation to the environment, and are often profane or obscene (*coprolalia*), or the imitations of sounds heard (*echolalia*). It usually appears during early adult life, but may develop in childhood. The patient becomes more or less melancholy, and may even be violently insane.

Paralysis Agitans.

This is characterized by a peculiar, fine tremor of the extremities, rigidity of the muscles, a characteristic attitude, disturbance of gait, and gradually progressive paresis. The disease usually occurs late in life, although cases have developed between the ages of twenty and thirty. The first symptom noticed is slight rigidity or impairment in agility of the arms. Later this rigidity involves all the muscles of the body, including those of the face, and there is a peculiar rigidity of the facial muscles, causing loss of expression, which is perhaps the most characteristic symptom of the disease. The rigidity also causes the patient to assume a typical attitude: the body and head are thrust forward, and the arms are slightly flexed and swing forward; if the patient stands, the knees and hips are slightly flexed, and the trunk is carried back for the purpose of balancing; the whole attitude, as well as all the movements, indicates stiffness. It will now be found that the patient will have difficulty in rolling over, if lying down, and that there is difficulty in commencing to walk and afterward a tendency to take quick steps (*festination*). The patient, if watched, will be seen to have from time to time a

slight movement forward or backward, which, if he is standing or walking, may cause him to fall in one direction or the other (*propulsion, retropulsion*). The symptom can often be elicited by giving the patient a slight push. Speech is also involved, difficulty in articulation being characterized at first by slight halting and then by the rapid utterance of the words. The tremor usually commences in the hands; it is spoken of as pill-roller's tremor (*q. v.*). The tremor of the head is a to-and-fro nodding movement. There may also be irregular movements of the toes or legs. The tremor is diminished or abolished temporarily by voluntary movement, and disappears during sleep. It is increased by excitement. In a few cases the tremor is entirely absent or occurs only at intervals (*paralysis agitans sine tremore*). Objective sensory disturbances are rare; the patients usually suffer from a persistent subjective sensation of heat, which is occasionally associated with an actual increase of the surface-temperature. The patients are excitable, in spite of the lack of facial expression, and may complain of insomnia. Trophic changes are rare, thickening of the skin is often present, and has been supposed to be the cause of the rigidity; but the skin is sometimes very delicate.

Tetany.

Tetany is probably an infectious disease characterized by cramp of the muscles of the arms and the persistence of peculiar nervous and mental alterations. The attack usually commences with paræsthesia or pain in the limbs; then the muscles controlling the fingers become stiff. The flexors gradually contract and draw the fingers and thumb together—the so-called obstetrical hand. The spasm is tonic in character, and may last for several minutes or even for many hours. It is often associated with intense pain. Nearly always it is bilateral. During the interval it may be reproduced by prolonged, severe pressure upon the nerve-trunks, particularly the median nerve (Trousseau's sign). The muscles show marked irritability to mechanical stimuli, particularly those of the face, and twitching may be caused by tapping upon the trunk of the facial nerve, upon the malar bone, or over the infra-orbital foramen (Chvostek's sign). The muscles show extreme electrical irritability, contract to very weak currents, and in some cases AOTe and COTe have been obtained (Erb's sign). Finally, the patient is extremely sensitive to the induced current (Hoffmann's sign). During the attack, and even during the interval, there is sometimes slight œdema of the face, hands, and feet, and the latter have a tendency to assume a partial equinovarus position. Often there is slight fever. Very rarely trophic changes occur, such as disturbance of the growth of the hair and nails or eruptions upon the skin. The disease appears to be endemic in certain localities, particularly in Vienna. It occurs most frequently in the months of February and March, and, curiously enough, has a special predilection for shoemakers. Tetany is also a symptom of certain morbid processes. Occurring after thyroidec-tomy, it often runs a severe course to a fatal termination. It may be present in myxœdema. It may also be associated with various disorders

of the gastro-intestinal tract in children, or with dilatation of the stomach in adults, and in this condition renders the prognosis very grave.

Occupation Neuroses.

These are characterized by the development of pain in the limb employed when the attempt is made to perform some habitual movement. They ordinarily occur in early adult life, particularly in neurotic individuals. In a few cases distinct hereditary influence can be traced. Great stress is laid upon the fact that the subject has been in the habit of performing the motions that cause the pain, such as writing in a faulty manner, holding the pen clumsily, and writing with the fingers alone. He first notices that he becomes more readily fatigued than usual, and there may be dull pains in the joints or in the palm of the hand. The painful sensations may then extend up the arm, often as far as the shoulder. They are rarely severe, but by their persistent, dull character are extremely annoying. The motor symptoms are characterized by a tonic spasm of the muscles employed in grasping the pen, so that it is held too tightly, and often there is difficulty in holding it properly. From time to time the spasmodic condition may increase and cause inaccurate strokes. The writing is usually heavy and often quite illegible. The muscles apparently never degenerate. The electrical reactions are normal or only slightly altered. If the patient learns to write with the left hand, the symptoms of the disease usually develop in it after a short time. Similar symptoms occur in piano-players, violin-players, dairy-maids, telegraphers, and various other persons who are obliged to perform the same type of movement for long periods.

Thomsen's Disease. (Myotonia Congenita.)

This is characterized by the occurrence of tonic spasm as the result of voluntary innervation of the muscles. It is described in Chapter XII., Diseases of the Muscles.

DISEASES OF THE SENSORY NEURON, WITH DISTURBANCES OF SENSATION.

These are generally included under the term *neuralgia*. Neuralgia is a condition characterized by pain of a dull, burning, or shooting character that occurs in the distribution of some particular sensory nerve or nerves. The pain may be remittent or intermittent and is often regularly paroxysmal. It is exaggerated, as a rule, by external irritation or emotional disturbance, and persists for some time after the exciting cause has ceased to act. During the attack the pain may extend beyond the limits of the particular nerve involved, and be felt in the surrounding or even remote parts of the body; this is called irradiation. The nerve-trunk is often tender, not only during the attack, but also during the interval, and there are special *tender points* along its course, usually where it passes through or over a bone. Associated symptoms are often present. The most

common are the vasomotor disturbances, the area of distribution of the affected side showing persistent or paroxysmal flushing or occasionally pallor, especially at the beginning of a paroxysm. Secretion of sweat is sometimes increased, and there may be exaggeration of the activity of glands supplied by the nerve. Occasionally there is marked œdema of the skin, and sometimes a herpetic eruption. Atrophy or occasionally even permanent thickening of the skin may occur. Very rarely in neuralgia there is local graying or falling out of the hair. Motor symptoms may also occur. These consist of spasmodic twitching that may be associated with exacerbations of the pain or more or less transient paresis. Neuralgias due to various general conditions sometimes have a characteristic localization. Thus in diabetes, sciatica occurs; in malaria, supra-orbital neuralgia; in neurasthenia, occipital neuralgia.

Special Forms of Neuralgia.

Neuralgia of the Trigeminal Nerve. This usually occurs in only one branch of the nerve, and is commonly unilateral. The terms ophthalmic, supramaxillary, and inframaxillary neuralgia are used to designate the special forms. Only one branch may be involved, as in supra-orbital neuralgia. The pain is paroxysmal and very severe, and is often referred by the patient to some supposed source of peripheral irritation, as disease of the nose, carious teeth, etc. It is usually associated with increase in the secretion of various glands, such as the tear-glands, the salivary glands, the nasal mucous membrane, etc. Trophic changes are not uncommon. These may vary from herpetic eruptions and graying of the hair to atrophy of the soft parts and even of the bones of the face. Occasionally trophic lesions also appear in the cornea.

Tic douloureux is a particular form that results apparently from disease of the Gasserian ganglion. The pain is usually intense, the paroxysms come on suddenly, last only for a few seconds, but may be repeated at very short intervals. During the attacks the features are rigid and the expression is one of intense agony.

Occipital Neuralgia. This involves the occipitalis major nerve, but occasionally the auricularis magnus and the nerves of the neck are also affected. The pain is distributed over the occipital region of the head, and is usually bilateral. The point of greatest tenderness is over the second cervical vertebra, usually slightly to one side of the spinous process. It may indicate caries of the upper cervical vertebræ.

Neuralgia of the phrenic nerve has been described, but its existence is exceedingly doubtful. The symptoms are said to be pain in the epigastrium, shooting to the shoulder, increasing when pressure is made in the epigastrium, with various respiratory disturbances—sneezing, coughing, etc. It is needless to say that these symptoms, should they occur, would be more likely to suggest an inflammatory process.

Brachial neuralgia is characterized by pain distributed in the arm of the affected side. This may be either persistent or paroxysmal. If the latter, paræsthesiæ in the hand or arm are frequent during the intervals. The points of tenderness are found where the nerves pass over

the bones or just behind the clavicle. Occasionally trophic changes are observed.

Intercostal neuralgia is characterized by pain distributed along the course of the intercostal nerves. There are three characteristic tender points—one next to the spinal column, one in the axillary line, and one over the sternum or rectus abdominalis. There are usually trophic disturbances in the skin over the affected nerve, characterized by reddening or especially by a herpetic eruption (*herpes zoster*).

Mastodynia, or painful breast, is a special form, characterized by pain, usually paroxysmal, and sometimes by the occurrence of painful nodules in the breast.

Lumbar neuralgia is characterized by pain radiating from the lumbar to the gluteal region. Occasionally the anterior surfaces of the thighs are also involved. The sensitive points are found over the lumbar vertebræ, along the edge of the crest of the ilium, and over the linea alba.

Crural neuralgia is characterized by pains radiating from the front of the thigh into the feet. Paræsthesiæ are frequently present during the intervals of the attacks.

Sciatica is characterized by pain in the posterior part of the thigh, often radiating to the feet. The disease usually occurs late in life, is more likely to affect men than women, and is frequently associated with obesity and with various gouty manifestations. It is an exceedingly common form, usually paroxysmal in character, the attacks being preceded by paræsthesiæ. The pain is increased by any movement tending to stretch the nerve, and as a result the patient walks with a peculiar gait, the thigh of the affected side being held fixed and parallel with the body, and the leg flexed on the thigh. This sometimes results in a slight curvature of the spine. According to Minor, the patient exhibits a peculiar method of getting up from the ground. He usually lies upon his back with the diseased limb flexed, gradually thrusts the body up with the hands, then lifts it on the hands and feet, the back being downward, turns half over toward the sound side, thrusts the trunk forward with the arm on the sound side, and gradually rises to an erect posture. The nerve is often sensitive throughout its entire length. The special points of tenderness are found near the posterior spine of the ilium, at the lower edge of the gluteus maximus, just outside the tuber ischii, and in the cavity of the knee-joint. The reflexes are usually slightly exaggerated. There are sometimes slight weakness of the muscles and occasionally fibrillary twitchings.

Other forms of neuralgia are *coccygodynia*, and various neuralgia-like pains in the viscera.

Meralgia paræsthetica is a disease somewhat similar to neuralgia. It is characterized by tingling, burning, or tearing in the area of the distribution of the external cutaneous nerve of the thigh, usually unequally bilateral, and made worse by prolonged exercise, either walking or standing. Frequently there is a tender point just below the anterior superior spine of the ilium. Sensory disturbances in the form of hypæsthesia,

hypalgesia, and diminished electrocutaneous sensibility are common. Stout persons and physicians appear to be particularly susceptible.

Acroparæsthesia is a condition characterized by tingling or pain in the extremities. The affected members are usually tender, and there is hyperæsthesia. Occasionally vasomotor disturbances are present. An allied condition is the symptom known as *tender toes*, which occurs in the course of typhoid fever.

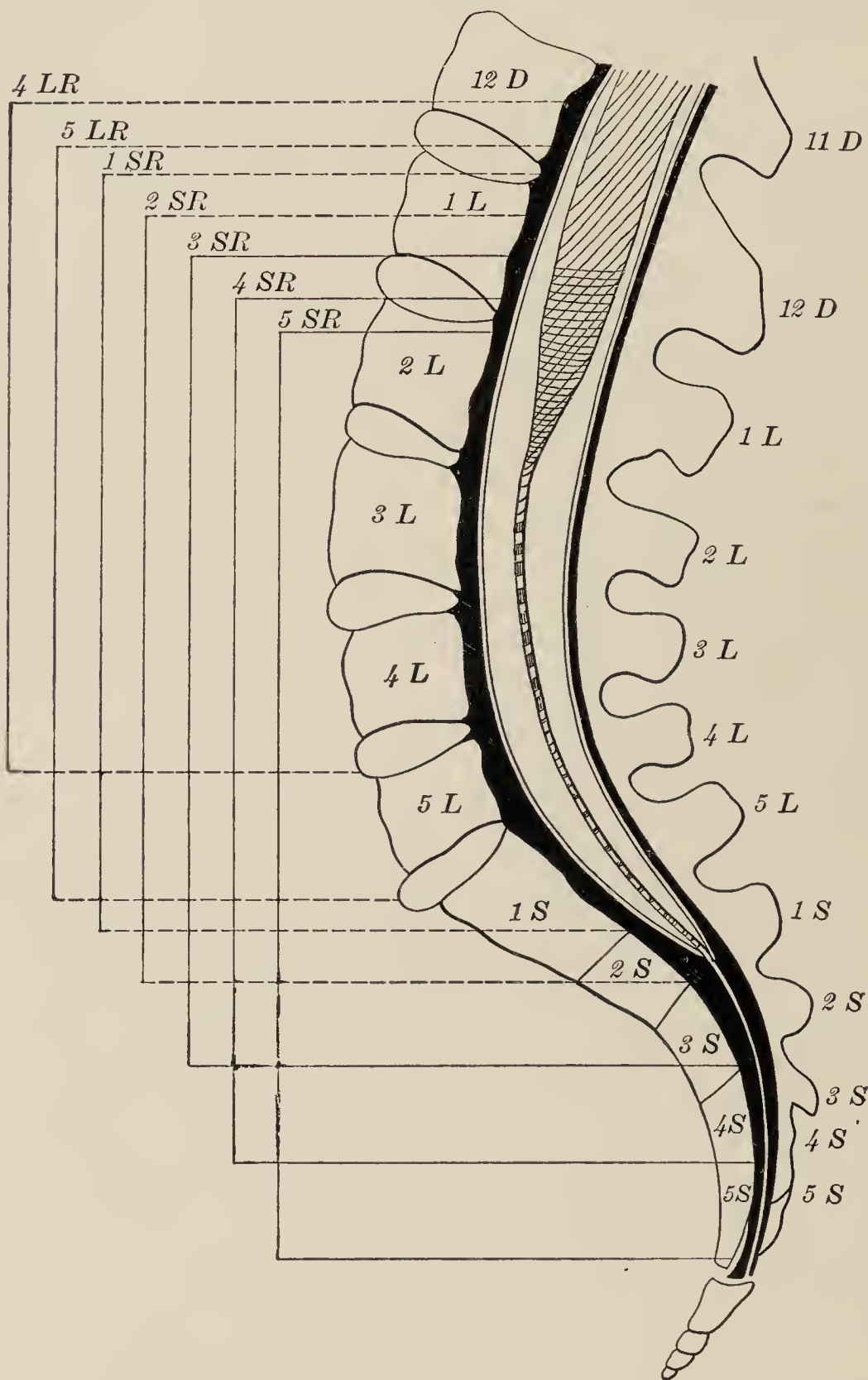
DISEASES OF THE SENSORY NEURON CHARACTERIZED BY DISTURBANCE OF MOTION AND SENSATION, AND BY TROPHIC DISORDERS.

Tabes Dorsalis. (Locomotor Ataxia, Posterior Spinal Sclerosis.)

This is characterized by ataxia, particularly of the lower extremities, lancinating pains in the legs, loss of the knee-jerk, and the presence of the Argyll-Robertson pupil. It usually occurs in the decennium from thirty to forty. A few cases have been reported that occurred before twenty, and some have occurred later in life. It affects men more commonly than women, and is exceedingly rare among negroes and the savage or semicivilized races in general; among the white civilized it is more common in the educated than in the ignorant. A history of syphilitic infection can often be obtained. It is divided into three stages: the pre-atactic, the atactic, and the paralytic. The symptoms of the *preatactic* stage frequently commence with disturbance in the nerves controlling the movements of the eyeball. There may be paresis of the abducens, giving rise to diplopia; of the levator palpebræ, giving rise to ptosis—both of which may be transient and may be discovered only by the history of diplopia or drooping of the eyelid. The reaction to light on the part of the pupil may be sluggish or absent, while the reaction to accommodation still persists (Argyll-Robertson pupil). The symptoms in the nerves of the lower extremities are particularly the lancinating pains that are felt in the posterior portion of the thigh. These are paroxysmal and the patient feels as if he had been stabbed. They are more frequent in damp weather, and are often confused with rheumatism. These *pains* may persist for years as an isolated symptom. The knee-jerk is absent (*Westphal's symptom*), and the patient may note that it is somewhat more difficult to walk in the dark. Often there is distinct weakness in the legs, manifested by fatigue after some moderate exertion and occasionally by giving way of the legs. The station in the early stage is usually only slightly affected. There is a sense of constriction about the body (girdle pain), and sometimes hypæsthesia of the lower extremities that may be associated with a slight hyperalgesia in the zone just above it. The patients may also remark that they have slight difficulty in urination and some diminution of sexual potency. The second stage, or the stage of *ataxia*, is characterized by the symptoms of the preceding stage, all of which are now pronounced. In addition the patient exhibits incoordination of movement, especially in the lower limbs. Station is so impaired that it is usually impossible for him to stand alone with the

eyes closed and the feet together (*Romberg's symptom*). Walking in the dark is difficult and results in frequent falls. In the daylight, with the aid of the eyes, the patient can usually walk quite well, but lifts the feet higher than usual from the ground, and separates them widely. (See

FIG. 390.

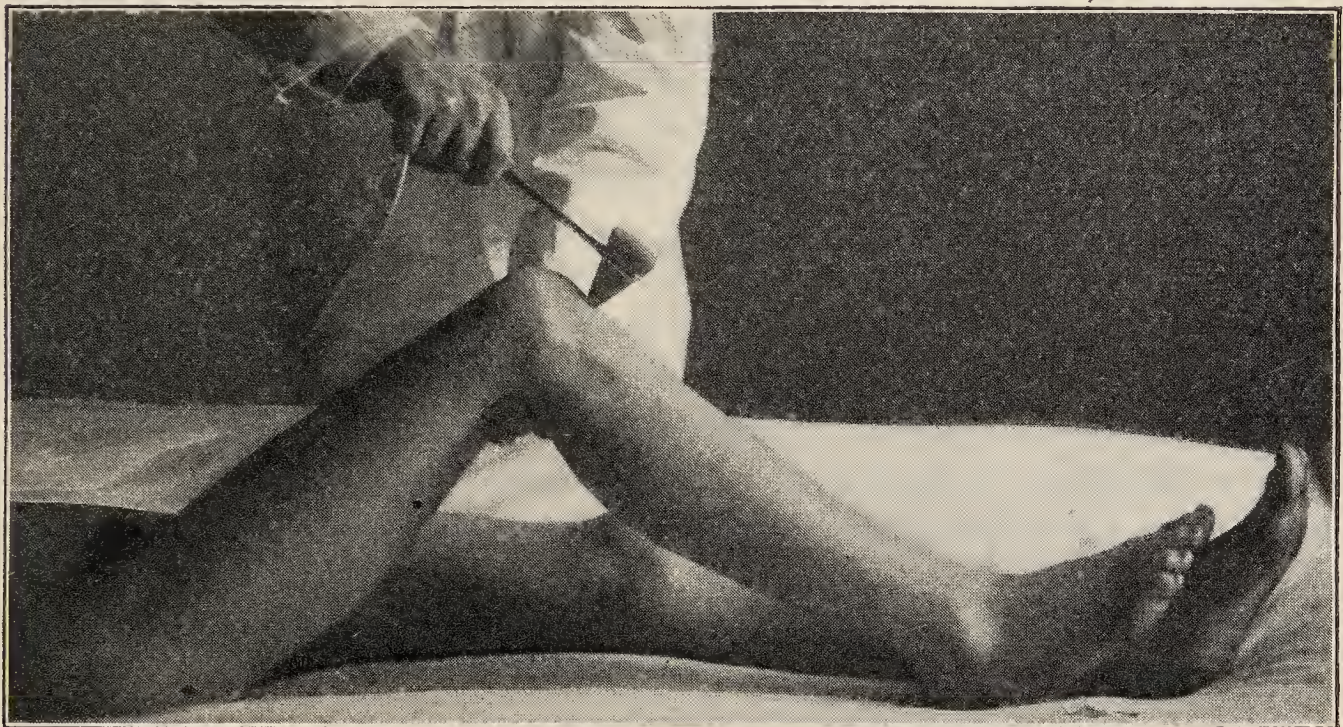


Schematic representation of the lower portion of the spinal cord and cauda equina, according to Raymond. The bodies and spines of the vertebræ are designated by the letters *D* (dorsal), *L* (lumbar), *S* (sacral), with the number of the vertebra prefixed. The roots are designated by the same symbols followed by *R*. The upper horizontal line indicates the level at which the root emerges from the cord, the lower horizontal line the level at which it pierces the dura. The vertical line joins the two horizontal lines belonging to the same root.

Ataxic Gait.) The incoordination is manifested by the difficulty with which the patients perform certain movements, such as touching some object with the tip of the finger—as, for example, the nose or ear—or in bringing the heel of one foot against the knee of the other. There is

diminished muscle-tone, so that it is possible to bring the foot to the shoulder without bending the knee; and, of course, absolute loss of the tendon-reflexes, even when reinforced. The nutrition of the muscles remains good, and the electrical reactions are not altered. There are paræsthesiæ, especially in the lower extremities; analgesia in the same situation, or sometimes delay in the conduction of pain. The nerve-trunks lose their sensitiveness to pressure; this is best tested with the ulnar nerve, which can be rolled under the finger against the lower end of the humerus without causing pain (*Biernacki's symptom*). Visceral anæsthesia is also present. The skin-reflexes are often preserved late in the course of the disease. Micturition is sometimes difficult; at other times there is incontinence, but insufficiency of the sphincter ani rarely occurs.

FIG. 391.



Testing the knee-jerk.

Impotence is complete. The Argyll-Robertson pupil is present; there are usually myosis, nyctalopia, and occasionally atrophy of the optic nerve, first manifested by disturbance of the color-perception (red and green blindness). In the latter condition it has been noted that when blindness has fully developed, the ataxia becomes less pronounced or may disappear completely (*amaurotic tabes*). The visceral crises are characterized by attacks of intense, cramp-like pain, involving usually the stomach, but sometimes affecting the larynx, heart, kidney, bladder, rectum, or other viscera. The laryngeal crises are often accompanied by distressing cough and dyspnoea that may simulate the symptoms of aneurism of the arch of the aorta. Trophic changes occur, of which the most common are the arthropathies. These involve particularly the knee, hip, and shoulder-joints. In addition, the patient may have painless falling out of the teeth or rapid softening of them. The bones often show a marked tendency to fracture, and an equally remarkable tendency to rapid knitting. Occasionally an osteo-arthropathy of the foot gives rise to the so-called tabetic foot, characterized by great thickening of the bones and a real or apparent shortening. In certain cases a chronic ulcer

develops on the sole of the foot, which usually progresses until it has produced perforation (*mal pérforant*). In the *paralytic* stage of ataxia the loss of muscle-tone has reached so extreme a degree that locomotion is impossible. The patients by this time have usually developed cystitis, and death occurs as a result either of exhaustion or of general septicæmia.

The Cervical Type of Tabes Dorsalis.

This is characterized by the development of the symptoms chiefly in the arms. The lightning pains occur in the upper extremities, there is loss of the bicipital and tricipital reflexes, and the girdle sensation is usually felt in the upper part of the thorax. The ocular symptoms are the same as in the usual form. The visceral crises are likely to affect the larynx. In this form ataxia in the legs, Romberg's symptom, and the absence of the knee-jerk may not be present until late in the disease, but there are usually disturbance of micturition and loss of sexual power.

Friedreich's Ataxia.

This is a disease of early life, distinctly hereditary in character, ordinarily affecting several members of the same family. It first appears between the ages of seven and twelve years. It is characterized by incoördination, loss of knee-jerk, weakness, irregular speech, and slight deformities. The first symptom is incoördination of the lower limbs. This gradually becomes more severe, the muscles grow weaker, the flexors more so than the extensors, often giving rise in time to pes equinovarus with hyperextension of the great toe. The muscles of the back also grow weaker, giving rise to scoliosis; the knee-jerks are absent, the pupillary reflexes remain normal, and intelligence is unaffected. The speech is peculiar, some of the syllables being pronounced readily and others slowly, with a drawl. The gait becomes markedly ataxic, the patients keeping the legs widely separated. In time the paresis and incoördination become so severe that walking is impossible. There are frequently irregular choreiform movements in the muscles and the so-called static ataxia—that is, inability to hold the limb in one position for more than a moment. There is often a nystagmus similar to that observed in multiple sclerosis. Sensation remains normal throughout. The course is progressive.

The *cerebellar* type of hereditary ataxia differs from the foregoing by the fact that the knee-jerks are exaggerated, and there is occasionally absence of the pupillary reflex to light.

DISEASES OF THE PERIPHERAL MOTOR AND SENSORY NEURONS.

These are all characterized by disturbances of motion and sensation, usually associated with more or less severe trophic changes.

Acute Neuritis.

Acute inflammation of the nerves is characterized by pain localized in the course and distribution of the nerve affected, paræsthesia, tenderness, and perhaps paresis or paralysis of certain groups of muscles. The pain is made more severe if the limb is held in such a position that the nerve is stretched. The nerve may be distinctly enlarged to touch. As it is a true inflammatory condition, there are usually constitutional disturbances, such as fever, malaise, etc. Often the disease is progressive, extending from the peripheral to the more central nerve-trunks. This is spoken of as ascending neuritis. Along the course of the nerve there are often vasomotor and secretory disturbances, such as pallor, redness, œdema, or the lesions may be more severe, such as atrophy of the skin, with glossiness of the skin, or trophic changes in the hair and nails, or even a herpetic eruption. The symptoms vary according as the nerve affected is chiefly motor or chiefly sensory. *Inflammation of a motor nerve* causes flaccid paralysis in the group of muscles supplied by the nerve, and if the inflammatory process persists, these muscles rapidly atrophy and give the reactions of degeneration. The reflexes in the parts controlled by these muscles are lost. The diagnosis can usually be made by a careful study of the distribution of the paralysis. The paralysis may be permanent, or recovery may ensue if the process has not proceeded too far. *Inflammation of a sensory nerve*, in addition to pain, is usually characterized in the early stages by hyperæsthesia of the skin in the distribution

FIG. 392.



Alcoholic polyneuritis. Foot-drop and wrist-drop.

of the nerve, which later becomes anæsthetic, and is often associated with hyperalgesia (*anæsthesia dolorosa*). Vasomotor and trophic changes are common. The course is usually favorable—that is, in time, either as a result of reparative processes in the nerve or by compensatory action of the adjacent nerves, the sensory disturbances disappear.

Multiple neuritis is characterized by the appearance of the symptoms of the disease in a number of nerves at the same time. The nerves of the limbs are far more frequently affected than those of the trunk. The symptoms are modified by the cause. In alcoholic polyneuritis there are

usually slight paræsthesiæ of the limbs, with marked paresis of the muscles, particularly the extensors, giving rise to foot-drop and wrist-drop. (See Fig. 392.) The disease usually affects all four extremities. In lead-poisoning the disease is sometimes unilateral, is usually restricted to the arms, and the sensory disturbances are very slight or absent. There is paralysis of the extensor muscles of the arm, which, in severe cases, goes on to muscular degeneration. Neuritis may also be produced by arsenic. Diphtheritic polyneuritis is usually characterized by paralysis of the muscles of the palate, but occasionally the muscles of the limbs are also involved. In certain of the chronic forms of polyneuritis, instead of or with the loss of power, there is marked loss of coördination. This is spoken of as the ataxic variety.

Beri-beri, or **kakke**, is an infectious disease of the Pacific Islands and the western coast of Asia, characterized by the symptoms of a peripheral multiple and symmetrical neuritis. It is considered in the chapter devoted to Infectious Diseases.

Multiple neuromata sometimes occur very extensively upon the nerves of the skin, at times producing symptoms of multiple pressure upon the nerves—that is, paræsthesiæ, paralyses, or loss of sensation. At other times they produce no symptoms whatever, and can be recognized only by inspection.

DISEASES OF THE SPINAL CORD INVOLVING THE CENTRAL MOTOR NEURONS.

Primary Spastic Paraplegia.

Primary spastic paraplegia is characterized by weakness of the legs without muscular degeneration and with increased reflexes. The disease was formerly supposed to be the result of the involvement of the lateral columns of the cord. The first symptom is weakness or a feeling of heaviness in the legs; then spontaneous cramps appear. The reflexes are greatly exaggerated, and the muscle-tone is so increased, particularly in the extensor muscles of the thigh and the muscles of the calf, that the patient walks with the leg partially extended, dragging the toe along the ground; the arms are rarely involved. The electrical reactions of the muscles are normal. The sphincters are rarely involved, and sensation usually is unimpaired. If cramps are frequent, however, the muscles may be sore. In children the adductors become stronger than the abductors, and a peculiar, cross-legged gait is thereby produced. It may occur in several members of the same family (*familiar form*), the symptoms usually appearing in early adult life. It may develop in early childhood, often after premature or difficult birth (*Little's disease*). In this form the gait is characteristic, the feet being lifted high from the ground to prevent dragging of the toes (stepper or chicken gait).

Amyotrophic Lateral Sclerosis.

The disease usually occurs in adults about middle life. In very rare cases it occurs in children. It is characterized by a spastic paraplegia,

with exaggeration of the reflexes and degeneration of the muscles. Spasms are common, and may give rise to muscular pains. The symptoms consist of weakness in the legs, which at the same time become stiff; the muscles rapidly atrophy; there are fibrillary twitchings and reactions of degeneration. The arms are usually involved first, the degeneration commencing in the muscles of the hands and giving rise ultimately to the production of various deformities, such as the claw-hand. The tendon-reflexes are greatly exaggerated; there are patellar and ankle clonus. The Babinski phenomenon is present. The muscles are greatly weakened, but remain rigid until late in the course of the disease. The sphincters are rarely involved, the pupillary reflexes are normal, and there are no sensory disturbances. Bulbar symptoms—that is, paralysis of the larynx, pharynx, and palate—occur, giving rise to dysphagia,

FIG. 393.



Testing the Babinski reflex.

dysarthria, and occasionally to entire loss of speech. In the early stages of this complication there may be distinct jaw clonus, elicited by tapping upon the lower jaw or the chin. When the bulbar symptoms become advanced, the lower part of the face is absolutely expressionless. It has a mask-like appearance, and often saliva dribbles from the mouth. In this stage inspiration pneumonia, due to involvement of the laryngeal branches of the vagus, is quite common and usually causes death.

Multiple Sclerosis.

This is a condition that involves the spinal cord and brain. The situation of the lesions is not constant, and therefore the symptomatology is variable. It usually commences at about the age of thirty years, and

is more common in men than in women. The characteristic symptoms are intention tremor, nystagmus, and scanning speech. The *intention tremor* involves chiefly the limbs, and causes disturbance of writing, walking, etc. It is most easily elicited by having the patient grasp some small object. There may also be rhythmical oscillations of the head. Ataxia, apart from the intention tremor, has also been described. There is usually persistent lateral nystagmus. The tendon-reflexes are greatly exaggerated, and there are ankle-clonus and a spastic gait. The pupillary reactions are normal. Occasionally diplopia occurs as a result of abducens paralysis; it may be transient. In a large proportion of the cases there is more or less complete atrophy of the optic nerve. The speech is slow, drawling, and often tremulous. The voice lacks expression, and, on account of a slight nasal tendency, is usually disagreeable. Not infrequently there may be stuttering. Sensory changes consist of paræsthesiæ, transient hyperæsthesiæ and anæsthesiæ, and occasionally disturbances of the function of the bladder. Less frequent symptoms are vertigo, occurring in paroxysmal attacks, diminution of intelligence, alternate states of depression and exaltation, and a tendency to laugh or weep without provocation. Apoplectiform and epileptiform attacks may also occur. The disease is usually chronic, but from time to time there are exacerbations. It appears to be frequently associated with hysterical manifestations. In some cases bulbar symptoms appear early and rapidly lead to death.

Hypertrophic Cervical Pachymeningitis.

This is characterized by pain in both arms, followed by muscular degeneration commencing in the hands. Later, there may be spastic paraplegia of the legs, with anæsthesia of the body below the affected segment. Occasionally this disease, which is usually due to tuberculous meningitis, may occur in other portions of the spinal cord, giving rise to various symptoms.

Acute Spinal Meningitis.

Acute spinal meningitis is characterized by intense pain in the back, radiating into the legs; rigidity of the spinal column, with opisthotonos; intense hyperæsthesia of the skin of the body, and, if the disease lasts long enough, paralysis. Kernig's symptom—that is, the inability to extend the flexed leg as a result of flexor cramp—is present. The *tâche spinale* occurs also in other conditions. It is always due to an infection, and therefore fever, chills, and prostration are present. The diagnosis is greatly assisted by examination of the fluid withdrawn by spinal puncture (*q. v.*).

Syphilitic Spinal Meningitis.

Syphilitic spinal meningitis produces a great variety of symptoms. There are, however, pains due to pressure upon the posterior roots, girdle pains of the body, and occasionally paralysis of the muscles of the extremities, with atrophy and degeneration. Often, also, the spinal cord is in-

volved, giving rise to the symptoms of pressure or transverse myelitis (*q. v.*) or Brown-Séquard's syndrome (*q. v.*). The sensory symptoms, aside from the pains, consist of hyperæsthesia, hypæsthesia, or anæsthesia. The tendon-reflexes of the lower extremities may be lost and reappear, and by some this is supposed to be pathognomonic of the disease.

DISEASES CHARACTERIZED BY THE SYNDROME OF TRANSVERSE INTERRUPTION OF THE SPINAL CORD.

Pott's Disease.

Pott's disease (*caries of the vertebræ*) in the majority of cases commences in childhood. Occasionally the first symptoms may be noticed in adult life. It is characterized by an angular deformity of the spine, spastic paraplegia, and various disturbances of sensation in the body below the level of the lesion. In the earlier stage the only symptom may be pain in the back, usually radiating around toward the ventral surface. There may be no deformity, but sudden pressure upon the head, jarring of the spine by coming down heavily upon the heels, and pressure over the tender point in the back may elicit sharp pains. In this stage there are usually slight exaggeration of the reflexes and perhaps a slight weakness of the legs. Later, the angular deformity becomes apparent, usually in the form of a sharp projection in the dorsal portion of the spinal column, but it may appear also in the cervical and lumbar region. The weakness of the lower extremities becomes more pronounced, and may give rise to an actual paraplegia. The pains are usually severe, radiate around the trunk, and sometimes affect other portions of the body. Sensation may be slightly impaired. There may be distinct dissociation below the lesion—that is, loss of temperature- and pain-senses, with preservation of tactile-sense—or there may be total anæsthesia. The bladder is usually involved, there is difficulty in micturition, and in the later stages, incontinence of urine or overflow. As in myelitis, bed-sores or other trophic changes of the skin are very likely to develop, and patients suffer severely in general nutrition. In the earlier stages, and more particularly in the stage of recovery, after the deformity has become stationary, ataxia may exist. The reflexes are sometimes greatly exaggerated, and there are often ankle-clonus and Babinski's phenomenon. When the paraplegia has become complete, all the reflexes are usually abolished. Girdle sensation is also very common. The tuberculin reaction, if positive, is not of much value in this disease, as other tuberculous foci may be present, although not suspected, and cause a rise of temperature after the injection. It is especially untrustworthy in adults. The course is very variable. At times the destruction of the body of the vertebra is rapid, and the symptoms develop acutely. At others, the condition occurs very slowly, and the symptoms, even after years' duration, may be exceedingly slight. Often after progressing to a certain extent the spinal deformity becomes stationary, and the nervous symptoms may gradually disappear. Caries of the upper cervical vertebræ produces pains that involve the neck and the occipital region of the head. The position of the head is peculiar: it is drawn slightly forward

and carried very rigidly, and the chin is elevated. These patients may sometimes die suddenly as a result of pressure by the odontoid process on the medulla.

Tumors of the Cord.

The symptoms of this condition are extremely variable, and depend upon the location, nature, and extent of the growth. Occasionally deformities occur as a result of pressure upon the arches of the vertebræ. Paraplegia usually develops, sometimes very suddenly, sometimes gradually. There are usually exaggeration of the reflexes and ankle-clonus; but these in time may disappear, or may never occur if the tumor is situated in the lumbar region. When the posterior roots are pressed upon, there are root-pains and the girdle sensation. Sensory disturbances are more or less complete according to the degree of destruction that has occurred in the spinal cord. At first there is ordinarily only hypæsthesia; later there may be dissociation of sensation, and finally, when the transverse lesion has become complete, total anæsthesia. After complete destruction of the spinal cord at any point trophic changes occur.

The diagnosis of the position of a tumor is more important than the recognition of its nature. The position may be at any level of the cord, and in the vertebræ, the meninges, or the substance of the cord. The segment or segments involved may be recognized by the determination of the upper level of the sensory and motor changes. If the anterior cornua or roots are destroyed, the muscles supplied by them will give the reactions of degeneration. If the posterior roots are irritated, neuralgic pains may be felt along the course of the nerves arising from them. If the tumor has produced a total transverse lesion, the symptoms at the different levels will correspond to those given in the table on pages 335 and 336. The situation of the tumor with reference to the spinal cord or the walls of the canal is more difficult to determine, and errors are not uncommon. Deformity of the spinal column is a valuable sign of vertebral tumor. Tenderness is of more doubtful significance, but is certainly more frequent when the bones are involved than when they are not. Root symptoms, preceding slowly developing compression, indicate a dural tumor, the predominance of motor or sensory changes, or the existence of a more or less well-developed Brown-Séquard syndrome (see page 337), suggesting that the tumor is on the anterior, posterior, or lateral aspect of the cord. Intramedullary tumors may give rise to symptoms resembling those of syringomyelia—that is, dissociation of sensation and the symptoms of transverse lesion. Naturally, the size and location of the tumor have considerable influence upon the early symptoms. If the tumor enlarges sufficiently to produce pressure, all the conduction-paths are interrupted. The nature of the tumor may sometimes be suspected by the course; if very slow, it is probably benign. Primary malignant tumors in other parts of the body strongly suggest metastatic growths.

Chronic Internal Meningitis.

This is usually characterized by pain that radiates into various portions of the body, particularly the limbs, and by more or less hyperæsthesia.

The motor symptoms consist of tremors, spasms, and occasionally, when the anterior roots are involved, paralyses, with muscular degeneration. In the milder forms the only motor symptoms may be incoördination of movement. Herpetic eruptions along the course of the nerves arising from the involved posterior roots are quite common. There is usually more or less rigidity of the spinal column, with pain on movement. The disease is usually syphilitic, and is often associated with tract degeneration in the spinal cord. Usually the posterior columns and the lateral columns are involved, and there may be the syndrome of combined sclerosis.

Acute Myelitis.

There are a number of varieties of this condition, the most common and typical being transverse myelitis. It is an acute inflammatory disease associated with constitutional disturbance—that is, chills, fever, and malaise—and is occasionally ushered in with a convulsion. The symptoms are those of transverse lesion of the spinal cord. Ordinarily the dorsal part is affected; and there are therefore in the earlier stages weakness and paræsthesia of the legs, and perhaps a girdle sensation and hyperæsthesia over the spine, in the zone supplied by the involved segment. Not infrequently painless spasms occur in the legs. In the course of a few days or hours the weakness of the legs increases until there is complete paraplegia. The tone of the muscles is enormously exaggerated, the knee-jerks are increased, and there are patellar and ankle-clonus, Babinski's sign, and often Sinkler's toe-jerk. The limbs are usually spastic and kept in a position of extension. From time to time the muscles give violent twitches. At first there is hypæsthesia, soon passing into complete anæsthesia up to the horizontal line surrounding the trunk, at which point there is girdle sensation, and above it there is a zone of hyperæsthesia. The muscles supplied by the affected segment atrophy and give reactions of degeneration. Those in the region below maintain their nutrition for a considerable time. There are difficulty in micturition, usually paralysis of the bladder, and finally overflow from retention. The urine becomes alkaline, cystitis develops very rapidly, and is often followed by extensive sloughing of the surrounding parts. Bed-sores occur early and extend deeply. Trophic lesions also occur in the legs, the skin becomes thin and glazed, and the toe-nails are brittle. Even arthropathies have occasionally been observed. After the acute stage has passed more or less improvement may occur, characterized by gradual return of power in the legs and partial recovery of sensation. The localization depends upon the same principles as in any other transverse lesion of the cord.

Acute Focal Myelitis.

This gives rise to only part of the symptoms described above, depending upon the tracts involved by the process and the various nuclei that have been destroyed. There is therefore usually a monoplegia, associated with exaggeration of the reflexes and irregular areas of anæsthesia, or,

if the focus be in the arm- or the leg-centre, diminution or loss of the reflexes and degeneration of the muscles.

Disseminated Myelitis.

Disseminated myelitis gives rise to a complicated group of symptoms, according to the number, situation, and extent of the lesions. It resembles perhaps most closely transverse myelitis (*q. v.*).

Chronic Myelitis.

Chronic myelitis is distinguished from the acute form by the more gradual development of the symptoms. The patient first notices weakness of the legs, perhaps characterized from time to time by complete transient loss of power (*giving way of the legs*). If the reflexes are examined at this time, they will be found slightly exaggerated; later they become very markedly increased, and ankle-clonus develops. The patient also complains, in the early stages, of paræsthesiæ in the limbs that may involve the arms as well as the legs, and sometimes the trunk. A girdle sensation is also frequently present. The sensory disturbances, however, are much less prominent than in the acute form. Total anæsthesia rarely occurs; usually there is only hypæsthesia, but as the different forms of sensation are unequally involved, there may be partial dissociation of sensation or anæsthesia dolorosa. Finally, muscular atrophies, and even severe trophic disturbances, occur, the picture ultimately resembling that of the late stage of acute myelitis.

Landry's Paralysis.

This is characterized by progressive paralysis of the legs, arms, and muscles of the throat, leading ultimately to death. The prodromes consist of slight fever, malaise, and paræsthesia in the legs. These are followed by weakness of the legs, which may involve both, or at first only one. This gradually ascends, and at the same time the paræsthesia becomes more distressing. There are, however, few or no objective sensory disturbances excepting occasionally a slight hypæsthesia. The reflexes are lost, the muscles are without tone, and the paralysis is therefore flaccid. Electrical changes do not occur, or only in very chronic cases. The paralysis gradually ascends, involving the muscles of the abdomen, the thorax, and the arms. When the thorax is involved, the patient usually has rapid respiration and complains of dyspnœa. Later there are symptoms of bulbar involvement, difficulty in deglutition, and interference with speech. The diaphragm becomes paralyzed, and the patient dies as a result of exhaustion. The intelligence remains normal throughout the disease; there is never loss of consciousness, and there is no disturbance of the function of the bladder or rectum.

Hemorrhage into the Cord. (Spinal Apoplexy.)

This is characterized by the sudden interruption of the function of the cord at a certain level. There is usually, at the time the hemor-

rhage occurs, severe pain, then rapidly developing paralysis of the legs, which may be flaccid if the lumbar region is involved, or spastic if the lesion is higher up. Hæmatomyelia into the cervical region may cause paralysis of the arms, but death usually occurs suddenly. The sensory disturbances are irregular in character. At times there is dissociation of sensation, more frequently complete anæsthesia up to the level of the hemorrhage. The patient has no fever, consciousness is not disturbed, but there is interference with the functions of the bladder and rectum. Occasionally the hemorrhage involves particularly one side of the cord or only one-half of the gray matter, producing the syndrome of Brown-Séquard (*q. v.*). The diagnosis can frequently be made from the subsequent course of the case. If death does not occur, rapid improvement is usually the rule. The sphincters regain their functions, power returns to the limbs, and ultimately the patient may recover completely. In some cases, however, recovery, although pronounced, is only partial.

Syringomyelia. (Cavity in the Spinal Cord.)

Although by some this is considered a congenital disease, the first obvious symptoms rarely occur before early adult life. These are of the segmental type, and in many of the cases the areas of sensory disturbances extend in the form of ribbons along the arms. Sometimes, however, they differ from this, and approximate more closely the cerebral type of anæsthesia. The anæsthetic areas on the trunk are always bounded by a horizontal line. Syringomyelia is characterized by a group of symptoms whose occurrence together is almost pathognomonic. First, dissociation of sensation: pain and temperature senses are lost, tactile and muscular senses are retained. Second, degenerative atrophy of the muscles, associated with fibrillary twitchings and alteration of the electrical reactions. Third, trophic lesions which may involve the skin, particularly that of the fingers or the joints. The size and position of the cavity and the extent of the destruction of the substance of the cord by the gliomatosis vary greatly, and as a result the symptoms, which are due entirely to the interference with the structures of the spinal cord, differ considerably in different cases. This difference, however, is quantitative rather than qualitative, and the three groups of symptoms are practically always present. The disease appears to develop with extreme slowness. The earliest symptom may be the occurrence of painless whitlows—that is, inflammation around the fingernail, with perhaps the ultimate destruction of the nail itself. These may recur in one finger after another for several years and without the presence of any other symptoms excepting perhaps a slight disturbance of sensation in the fingers. Later, muscular atrophies appear. These involve particularly the muscles of the shoulder or the hand. In the latter situation they may give rise to the appearance that occurs in progressive spinal muscular atrophy. At the same time the sensory disturbances become more pronounced, gradually ascending the arm and perhaps involving the trunk. The upper border forms a horizontal line about the body—that is, the alterations are segmental in type. The trophic changes may then

assume a more severe form, giving rise to deep, painless ulcerations in the fingers, and perhaps loss of the terminal phalanges. For a long time the symptoms may remain almost exclusively unilateral, and it is rare for the two sides to be affected equally. The motor symptoms, aside from the weakness resulting from the muscular atrophy, consist of weakness of the legs with exaggeration of the reflexes—that is, spastic paraparesis. At times the lower portion of the cord is particularly affected, and then sensory and trophic changes are found in the legs. Station may be slightly altered in the later stages of the disease, but this is by no means a characteristic symptom. Ultimately the patient develops scoliosis, trophic changes affect parts other than the hands, giving rise to arthropathies or to a form of dry arthritis with absorption of the bone. There may be vasomotor disturbances, and in some cases inequality of the pupils. The intellect is undisturbed. The patients ordinarily die as a result of exhaustion or pulmonary involvement, but occasionally in the later stages of the disease bulbar symptoms occur.

Morvan's Disease.

This is characterized by the appearance of painless whitlows in the fingers, sometimes associated with deep ulcerations of the soft parts. There are usually sensory disturbances similar to those found in syringomyelia, with the addition of tactile anæsthesia, but muscular atrophy rarely exists. The disease is exceedingly chronic. It is possibly only a variety of syringomyelia.

Traumatism of the Spinal Cord.

This may either produce destruction, partial or complete, of the tissue of the cord itself, giving rise to the syndrome of transverse interruption, or else give rise to a group of indefinite motor, sensory, and mental disturbances that have been grouped under the term traumatic neuroses. (See Hysteria.) The symptoms which result from an organic lesion may come on gradually or immediately. They are similar to those produced by pressure upon the cord or by hemorrhage into it.

DISEASES OF THE BRAIN CHARACTERIZED BY GENERAL SYMPTOMS AND SENSORY AND MOTOR DISTURBANCES.

DISEASES CHARACTERIZED BY MENTAL, MOTOR, SENSORY, AND SOMETIMES TROPHIC DISORDERS.

External or Pachymeningitis.

External or pachymeningitis is a rare condition, usually secondary to traumatism or abscess, characterized by fever, headache, often sharply localized, and convulsions. Frequently the symptoms are masked by the existence of some graver condition, such as status epilepticus, coma, or uræmia. Occasionally no symptoms are produced. If there is much thickening of the membrane, evidence of focal disease in the form of

paralysis or convulsions of the Jacksonian type may be present. *Hæmatoma* of the dura mater is a condition usually occurring in cases of chronic disease. There may be slight fever and headache without other symptoms. In some cases, however, the onset is sudden and apoplectiform in type. The patients develop hemiplegia, unconsciousness, and occasionally unilateral convulsions.

Internal or Leptomeningitis.

There are many varieties of this ; thus, according to the location, there may be meningitis of the convexity, of the base, or of the brain and cord ; according to the cause there may be suppurative, epidemic, or tuberculous meningitis ; other varieties not so easily classified are serous meningitis and meningismus.

Suppurative meningitis. The symptoms vary according to the nature of the process, its localization, and extent. The patient may complain of malaise and headache for a few days preceding an attack ; then there is often a chill, followed by fever, convulsions, and delirium. The headache becomes more and more intense, and frequently there is vomiting, sometimes with, sometimes without, nausea. The headache is often localized to the frontal or occipital regions ; occasionally, however, it is more general. From time to time there are acute exacerbations, causing the patient, especially if a child, to cry out—the meningitic cry. The skin is hyperæsthetic ; all the sensory nerves have their functions increased ; there are photophobia and inability to tolerate noises. Frequently there is paresis of the vasomotors of the skin, so that localized cutaneous irritation, such as may be produced by drawing the end of a blunt object across the surface, gives rise to a persistent red mark (*tache cérébrale*). Herpes labialis is very common ; less so is a disseminated erythematous eruption or irregular bluish patches ; the latter occur most frequently when there is violent septic infection. The patient usually lies with the head drawn far back and the muscles of the neck tense and rigid. This, however, occurs only when the cervical portion of the spinal cord is also involved. It is an exceedingly important and an almost pathognomonic symptom. Any attempt to straighten the head causes intense pain. Examination of the eye-grounds usually shows intense congestion and more or less perineuritis. Sometimes there is very distinct choked disk. The pupils are often unequal, and strabismus and even nystagmus frequently occur. Paralysis of any of the cranial nerves may occur, but is not so common as in tuberculous meningitis. The oculomotor or some of its branches is most frequently involved, although slight paresis of the facial nerve is not uncommon, and occasionally there is distinct monoplegia or hemiplegia. The tendon-reflexes are usually somewhat exaggerated, the muscular tone is increased, and Kernig's sign is usually present, although it can not be regarded as pathognomonic. Fever, headache, and delirium usually persist throughout the course of the disease ; the fever is often very high. In the early stages the pulse is usually relatively slow ; later it may become rapid. The blood-pressure is always increased. By means of Quincke's

lumbar puncture it is sometimes possible to make a bacteriological diagnosis from the fluid withdrawn. Meningitis due to pyogenic micro-organisms, such as the pneumococcus, staphylococcus, etc., may be suspected: when the fever is high; when there is marked retraction of the head, indicating spinal involvement; when there is general leucocytosis; when there is an excess of albumin (more than 1 per cent.) and of polymorphonuclear cells in the spinal fluid; and especially when there is or recently has been some other focus of pyogenic infection in the body, such as pneumonia, sepsis, or middle-ear disease. The course is progressive and almost invariably terminates in death. But remissions may occur or there may be considerable temporary improvement after spinal puncture.

Epidemic cerebrospinal meningitis may exactly simulate the symptoms of purulent meningitis. In some cases, however, the course is more prolonged, and even when the termination is fatal, there is apt to be a remission of longer or shorter duration. The diagnosis can be made only by the discovery of the meningococcus in the spinal fluid or the nasal secretion. If an epidemic exists, it may be readily suspected, but it must be remembered that other forms of meningitis are not less frequent.

Tuberculous meningitis is the most common form. It affects chiefly the base of the brain. There is usually recognizable tuberculosis of some other part of the body, and often a history of injury to the head. The course is slow, although in rare instances it may be very rapid; the prodromal stage is prolonged and the symptoms are slight, consisting of headache, insomnia, malaise, loss of appetite, progressive emaciation, and occasionally distinct psychical disturbance, such as extreme irritability, light coma, a confusional state, or loss of interest in the surroundings. As the disease progresses, more distinct lesions occur, the cranial nerves are involved, and there may be ptosis, diplopia, or paralysis of the pupil. Paresis or even paralysis of the facial nerve is common, and lesions of the other nerves may develop. Occasionally monoplegia or hemiplegia occurs. The fever is moderate, the course deliberate, and extreme intracerebral pressure develops rather late, so that the blood-pressure may not be greatly elevated. The fluid obtained by spinal puncture is usually clear, but tubercle bacilli may sometimes be obtained by centrifugation. It is said to contain an excess of mononuclear cells, but it is dangerous to base a diagnosis upon this sign alone. There is moderate leucocytosis (10,000 to 15,000), Kernig's sign is more frequently absent than present, the knee-jerks are usually increased, sometimes unequally, and ankle-clonus may occur. Spinal symptoms, such as rigidity of the spine and retraction of the head, are comparatively rare. Aside from the discovery of tubercle bacilli in the spinal fluid the only pathognomonic sign is the presence of tubercles upon the choroid. In very rare instances a secondary infection with some other form of micro-organism occurs in tuberculous meningitis. This appears to render the course shorter and more violent.

Serous meningitis is characterized by a marked increase of the intracerebral pressure, and the symptoms that result from it—headache, coma, or convulsions. There are usually slight fever, moderate leucocytosis,

increase in the reflexes, and slight rigidity of the back of the neck. The face is flushed, but eruptions are rare. The differential diagnosis from suppurative meningitis can be certainly made only by an examination of the spinal fluid. This contains more albumin and cells, chiefly ependymal, than normal, but is sterile. This condition usually occurs in the course of some infectious process.

Meningismus is a term invented by French clinicians to describe a condition that may occur in the course of severe infections; it is characterized by symptoms practically identical with those of serous meningitis, but more transient. Ordinarily it is entirely relieved by spinal puncture. As Kernig's sign may occur in acute infections, it is of little value in the diagnosis of this condition.

Cerebral Hemorrhage. (Apoplexy.)

This usually occurs in advanced life in patients who have pronounced arteriosclerosis. Occasionally, however, it occurs in young adults and in children. It is characterized by a great variety of symptoms, depending largely upon the location of the lesion. They may be divided into those

FIG. 394.

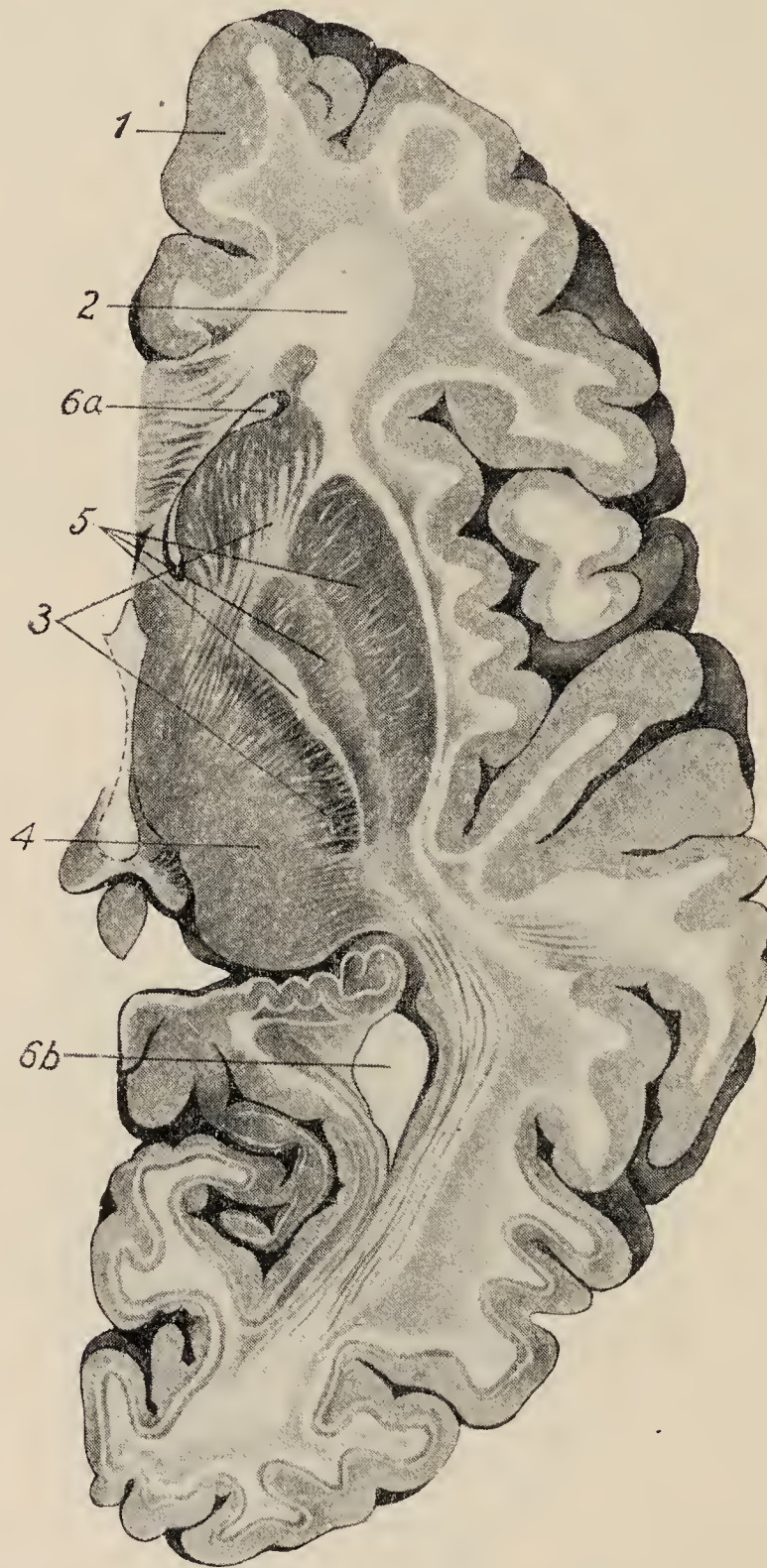


Vertical transverse section through the right hemisphere: 1, cortex; 2, white substance; 3, internal capsule; 4, optic thalamus; 5, lenticular nucleus; 6, part of the lateral sinus. (After DÉJÉRINE.)

of the attack and those of the post-apoplectic stage. The symptoms of the attack consist of prodromata—that is, headache, tendency to vertigo, a sense of fulness in the head, roaring in the ears, and perhaps some thickness of speech. These may pass off without further disturbance or may lead directly to an attack. The latter is usually characterized by

the sudden occurrence of complete unconsciousness. The patient falls to the ground, and there is at first a temporary pallor. This is succeeded by flushing of the face, which may become almost purple. The pulse is full, bounding, and compressed with difficulty. The blood-pressure is greatly increased. The breathing is stertorous, slow, and occasionally of the Cheyne-Stokes type; the pupils are usually contracted and often

FIG. 395.



Horizontal section through the right hemisphere; 1, cortex; 2, white substance; 3, internal capsule; 4, optic thalamus; 5, lenticular nucleus; 6a, anterior horn, and 6b, posterior horn of the lateral ventricle. (After DÉJÉRINE.)

unequal. Often there is vomiting or involuntary micturition or defecation. The limbs remain completely paralyzed, or in some cases there are unilateral convulsions. If, as is commonly the case, the hemorrhage has involved the motor tract, there is complete flaccid paralysis of one side, with lost reflexes. If death does not occur in the course of the first twenty-four hours, the patient usually begins to show signs of

consciousness, and may be aroused from his comatose condition by sharp questioning. He then may pass into a still more deeply comatose condition, with rise of temperature, followed by death, or there may be no further indications of hemorrhage, and recovery may set in. As a rule, in those cases in which the prognosis is favorable no rise of temperature occurs. It may now be found that the patient has hemianopsia, usually the visual fields on the same side of the lesion being blinded. Conjugate deviation may or may not have existed from the first, the patient ordinarily looking toward the sound side. If the speech-centre has been involved, there is absolute aphasia; but even when it is not directly affected, partial aphasia is very common. The hemiplegic limbs remain paralyzed; the others regain their power. It is now necessary to determine the extent of the damage and to locate as nearly as possible the position of the lesion. Complete hemiplegia may involve the lower branch of the facial, the arm, and the leg. The upper branch of the facial and the muscles of the trunk commonly escape, although the former may show slight paresis. Often there is considerable inequality in the paralysis of the different parts. The arm may be more involved than the leg, or the reverse, or the face may escape almost entirely. Sensory disturbances may or may not be present. There is sometimes loss of all forms of sensation and sometimes disturbance of only the tactile or the muscular sense. Occasionally, when tactile sense is preserved, there may be loss of the stereognostic sense. Complete hemiplegia with disturbance of sensation almost invariably indicates destruction of the internal capsule upon the opposite side. Motor disturbances in the form of clonic convulsions may also occur in the paralyzed limbs, and occasionally, probably as the result of a double lesion, in the limbs of the sound side. They are commonly the result of cortical lesion, apparently irritative in character—either infarction or else some growth pressing upon and involving the cortex. As the case progresses there are usually more or less return of motor power and almost complete return of sensation. This may, however, be exceedingly gradual, several weeks elapsing before the sensory disturbances have entirely disappeared. The muscles that remain permanently paralyzed gradually atrophy, but nearly always give normal qualitative electrical reactions until the muscular substance disappears, leaving contracted fibrous tissue. The muscles themselves may contract early, the flexors ordinarily overcoming the extensors. Repeated attacks of apoplexy are by no means uncommon, and the double lesions thus produced may give rise to very complex symptom-groups. Hemorrhage into the pons or medulla gives rise to various forms of crossed paralysis. (See also *Cerebral Localization and Aphasia*.)

Cerebral Embolism and Thrombosis. (Softening of the Brain ; Encephalomalacia.)

This condition may occur at any period of life, and is nearly always associated with some valvular disease of the heart or an extreme cachexia or anæmia predisposing to thrombosis. It is characterized by symptoms very similar to those of cerebral hemorrhage. Prodromal symptoms, in

the form of headache, vertigo, weakness, and malaise, are often present. At times there may be also slight impairment of speech, or the patient may be dull and apathetic. The attack usually comes on more gradually than hemorrhage, although this is not invariably the case. In some instances consciousness is not entirely lost, and as a result the hemiplegia may develop before the coma. When unconsciousness does occur, there are usually less congestion of the face and not such marked evidence of increased arterial tension as we find in hemorrhage. Among the other general symptoms may be mentioned convulsions, often epileptiform in type, vomiting, and occasionally delirium. The permanent symptoms resemble exactly those produced by hemorrhage, but recovery is usually more rapid and more complete than in the former condition. As the Sylvian artery is most frequently affected, the symptoms resemble the cortical type of apoplexy.

Apoplexy occurring in children differs from that occurring in adults only by the fact that the initial symptoms are more severe, and the convulsions are frequent and may be repeated. The permanent symptoms differ slightly, inasmuch as aphasia rarely persists. The paralysis may be partial, and may in some instances be replaced by or associated with athetoid movements. Sensation is rarely impaired.

Bulbar Paralysis.

Bulbar paralysis is a disease of the peripheral motor neurons arising in the medulla. It is characterized by the degeneration of the muscles of the lips, tongue, and pharynx, which grow smaller, exhibit fibrillary twitchings, and give the electrical reactions of degeneration. Sensation is not affected. The course is slowly progressive. The earliest symptom is dysarthria, then difficulty in swallowing, chewing, and phonation. The face becomes expressionless, the mouth remains open, saliva dribbles from it, and occasionally the eyelids are involved and the eye remains open (lagophthalmos). The cardiac action and respiration may be rapid. Death usually occurs as a result of inspiration pneumonia or exhaustion. In the variety known as *asthenic bulbar paralysis* there may be long remissions or even permanent recovery. The muscles become readily fatigued and exhibit the myasthenic reactions.

Pseudobulbar palsy is characterized by the same symptoms as bulbar palsy, but no lesions are found in the medulla; symmetrical lesions are sometimes found in the cerebral hemispheres.

Acute Encephalitis.

This is a condition that rarely can be diagnosticated during life. It may be suspected, however, if, in the course of some other acute infectious disease, especially acute endocarditis, the patient develops intense headache, severe delirium, and perhaps local palsies. There may be general exaggeration of all the reflexes, with ankle-clonus, and usually hyperæsthesia of the skin and exaltation of the special senses. Examination of the eye-grounds usually fails to reveal optic neuritis. There is leucocytosis, and cultures from the blood and spinal fluid are often positive.

Abscess of the Brain.

This is usually secondary to some local focus of suppuration or to pyæmia. There is often a history of mastoid disease. It may be acute or chronic. Acute abscesses are characterized by violent general disturbances, chiefly fever, chills, leucocytosis, headache, delirium, and coma. Chronic abscesses give rise to symptoms of local disease, which depend, of course, upon the location of the abscess. The course is usually intermittently progressive; thus there may be first a monoplegia, then hemiplegia, and finally aphasia; or the earliest symptom may be hemianopsia, followed by paralysis. The commonest seat is in the temporo-sphenoidal lobe, as a result of infection following ear disease. This often gives rise to mind-blindness or amnesia. Sometimes there are no general symptoms if the abscess is located in the so-called blind regions of the brain. The focal symptoms may not be manifest until rupture has occurred. This often gives rise to an epileptiform attack. Chronic abscess of the brain is occasionally observed in cases of congenital heart disease.

Tumors of the Brain.

Like the preceding lesion, these give rise to two groups of symptoms: general, which are merely those of increased intracranial pressure; or local, which are due to involvement of centres and tracts. The general symptoms of brain tumor are: (1) Headache; this is usually very severe, of a boring character, and subject to exacerbations. (2) Vomiting; this is paroxysmal, and often occurs without nausea. (3) Papillitis; it usually occurs early, is intense, and often leads rapidly to blindness. (4) Increased blood-pressure. The local symptoms are, of course, numerous. Tumors in the *frontal lobe* may give rise to none, or only to slight disturbance of intelligence and perhaps to a tendency to make puns. The headache is usually frontal, and occasionally in subcortical tumors there may be nystagmus. Tumors in the *motor region* may cause irritative or destructive changes in the tissue. Irritation is manifested by local spasms, which may or may not be succeeded by general convulsions (Jacksonian epilepsy). Paralytic lesions belong to the monoplegic or hemiplegic types. Tumors in the *parietal lobe* may cause interference with the muscle-sense or some disturbance of vision- or speech-centre, according to their situation. The loss of the stereognostic sense is a common symptom. Tumors in the *occipital lobe* usually cause mind-blindness—that is, inability to recognize objects, and preservation of the pupillary reflexes. If unilateral, they often cause hemianopsia. Tumors in the *cerebellum* usually produce marked disturbance in coördination, and the patient exhibits the peculiar staggering gait, not increased by closing the eyes, that has been described. Optic neuritis is an exceedingly common complication, but there may be descending atrophy of the optic nerve without preceding choked disk. The headache is severe; it is situated in the posterior portion of the head; there is often tenderness over the external occipital protuberance. The patient may suffer from vertigo, which at times is almost constant. The general symptoms are usually pronounced. There

are convulsions, vomiting, disturbances of respiration, and sometimes marked slowing of the pulse. Tumors of the *basal ganglia* produce very variable symptoms, the majority apparently being the result of pressure upon the internal capsule. Tumors of the *thalamus* often give rise to hemianæsthesia with loss of mimicry, and in some cases apparently to athetoid movements. In all forms of cerebral tumors, but particularly in slowly growing tumors of the cerebellum, the early symptoms may be those of neurasthenia or even hysteria, and the diagnosis for a long time is exceedingly difficult. Tumors in the different fossæ of the skull often give rise to symptoms dependent upon pressure upon the cranial nerves. In the *anterior fossa* there may be loss of the power to smell upon one side. In the *middle fossa* the nerves chiefly affected are the optic, giving rise to unilateral blindness; or, if the tumor involve the chiasm, to bitemporal hemianopsia; if it passes upon the oculomotor nerves, the abducens, and the pathetic, it may give rise to more or less complete ophthalmoplegia. Tumors in the *posterior fossa* commonly involve the facial and auditory nerves; and facial paralysis with nerve-deafness on the same side is characteristic of tumor in this situation. The hypoglossal nerve may also be involved. Tumors may grow slowly, rapidly, or cease to increase in size, and the symptoms show a corresponding rate of development. In rapidly growing tumors apoplecticiform attacks are frequent, but a certain amount of compensation occurs, and remissions are not uncommon. In slowly growing tumors the symptoms may remain apparently stationary for long periods. Tumors are sometimes entirely latent, and are only discovered accidentally at the autopsy.

Sclerosis of the Brain.

This is usually a diffuse or a multiple lesion that gives rise to a great variety of symptoms, more or less indefinite in character. Ordinarily the lesion is congenital or develops shortly after birth. The patient remains an imbecile or an idiot, and soon develops epileptic convulsions. If the sclerosis is more pronounced on one side than on the other, there is usually a tendency to fall toward the opposite side. There may be arrest in development in these limbs, and more or less muscular paralysis. Occasionally, apparently as a result of fœtal thrombosis or embolism, the sclerosis may be limited to one portion of the brain or even to one hemisphere. In this case there is always arrest in the growth of the opposite side of the body.

Hydrocephalus.

Hydrocephalus (*chronic infantile form*). This is characterized by an extraordinary alteration in the contour of the head, which becomes greatly enlarged and globular in shape, while the face remains small and infantile in appearance. The fontanelles and sutures remain widely open, or are closed only very late by Wormian bones. The veins of the scalp are very distinct, and occasionally fluctuation may be detected in the head. The symptoms are sometimes exceedingly pronounced; at other times entirely absent. Persons with a moderate degree of hydrocephalus have

displayed through life a normal intelligence. In pronounced cases the head is heavy and the muscles of the neck are unable to support it. The child develops slowly in intelligence or may even be an imbecile or an idiot, and epileptic convulsions are very common. Occasionally ocular symptoms may be present. These consist of ptosis, strabismus, or nystagmus, and sometimes of atrophy of the optic nerve and blindness.

Acute Delirium.

This is a disease characterized by prodromata and a stage of excitation, and usually terminates in death. The prodromata consist of disturbances of the general health, loss of appetite, and insomnia. The patient is restless, anxious, and may show diminution of intelligence, and become more or less violent. He then rapidly passes into the stage of excitation, is restless, noisy, and frequently homicidal, shouting disconnected words or sentences, singing or shrieking. Sometimes there are delusions of persecution, and he attempts to escape. In addition, there are the symptoms of the so-called typhoid state—high fever, profound prostration, dry tongue, and rapid and weak pulse. The patient refuses all food, is continually active, and emaciates very rapidly. Among the objective symptoms are increase of the reflexes, narrowing of the pupils, and hyperæsthesia, with more or less hypalgia. From this stage the patient passes into a state of collapse, lies in a condition of muttering delirium, with carphology, and usually dies from exhaustion.

General Paralysis of the Insane.

General paralysis of the insane is a form of progressive dementia characterized by delusions of grandeur or states of depression associated with exacerbations of maniacal character. There are, in addition, weakness and tremors of the muscles of the face, paresis of the extremities, Argyll-Robertson pupil, and peculiar disturbances of speech. It is a disease of middle adult life, the first symptoms occurring between the thirtieth and fortieth years. There is often a history of syphilis. It is usual to recognize three stages: The prodromal stage, characterized by irritability or sometimes by depression; diminution or loss of the moral sense; impaired judgment, particularly in business affairs; and a tendency to extravagance and dissipation. Frequently symptoms associated with degeneration, such as intolerance for alcohol, intense egotism, etc., appear. The sexual appetite in this stage is often greatly increased. Memory fails, and the intellectual capacity is considerably diminished. There are often slight disturbances of speech, and sometimes paralytic pupils. Frequently there are insomnia and occasional attacks of migraine. In the second stage, which usually develops gradually, the attacks of migraine are replaced by apoplectic or epileptic attacks or by distinct maniacal conditions; memory is greatly impaired, the intellect is considerably disturbed, the patient becoming unable to do easy mathematical problems, to comprehend his environment, or to sustain a simple conversation. Usually there are delusions of grandeur, the patient believing himself rich, beautiful,

successful, intelligent, and reiterating constantly his advantages, although from time to time there are states of depression and partial recognition of the failure of power. In other cases, however, particularly among chronic alcoholics, there is distinct melancholia; the patient is hypochondriacal, or may have delusions of persecution or a sense of misfortune. The disturbances of speech are characteristic; the most common is the omission of syllables. This may best be tested by asking the patient to repeat certain words, particularly those containing a number of r's and l's, as "third artillery brigade," "truly rural," etc. The intonation is often monotonous and often there is an unpleasant nasal drawl. There is marked tremor of the lips and of the tongue, producing a sort of ataxia in the speech, with the disturbance of the formation of nearly all the sounds. The pupillary changes are similar to those described in the prodromal stage, but usually are more pronounced. Piltz and Westphal have described a peculiar reflex contracture of the pupil upon forcible closure of the lids that is more common in general paresis than in any other disease. The extremities are weak, and often exhibit distinct tremors. Trophic lesions are not so common as in tabes, but do occur. They are of the same character and possibly due to tabetic changes in the cord. Finally, the patient becomes completely demented, usually lies quietly and placidly in bed, or occasionally mutters unintelligible sounds. Sensation, either as a result of impaired perception or because of degenerative changes in the peripheral nervous system or the spinal cord, becomes greatly impaired, particularly the pain sense. The patient is unable to stand, and has involuntary or rather unperceived micturition and defecation, and frequently develops bed-sores or cystitis; even rupture of the bladder may occur. A curious and quite common symptom is the gnashing of the teeth, which in some cases is almost persistent. Death usually occurs from exhaustion. Among the less frequent symptoms are a curious unsteadiness of gait, exaggeration of the reflexes, and rapid diminution in weight, particularly in the last two stages.

Epilepsy.

This is a condition characterized by attacks of clonic convulsions associated with loss of consciousness and usually some impairment of intelligence. In the characteristic epileptic fit we can usually distinguish three stages: the prodromal stage, the attack, and the post-epileptic stage. In the prodromal stage auræ are frequently present. These may be of the most varying character. A patient may have either a curious sensation in the epigastrium, paræsthesiæ in a limb, and the subjective sensation of movement, or disturbance of the special senses, particularly an unpleasant odor or a whirring sound. Sometimes the sensations are painful or distressing, as a sense of constriction about the throat. At other times there is giddiness, vertigo, or nausea, or the recurrence of some particular idea. Occasionally the auræ consist of some imperative movement, such as whirling about, running, or jumping. At the commencement of the attack there is usually a cry—the epileptic cry. Ordinarily this is a curious sort of gasping, due to the forcible contraction of the thorax and partial

closure of the glottis. In some cases, however, it may be a loud shriek. The patient then falls to the ground, and the convulsive movements commence. These are rarely of equal vigor on both sides; the head and the eyes show conjugate deviation; the face is bluish and pallid; the mouth is filled with frothy fluid, which is often blood-stained because the tongue has been bitten; the limbs may be extended or flexed in tonic contraction. This is soon replaced by a violent to-and-fro tremor. The patient is completely unconscious, and may, in falling, cause himself serious injury. There is no conjunctival reflex; the pupils are widely dilated; frequently the urine is passed during the attack, and there is occasionally profuse sweating. Toward the end the convulsive movements become less frequent. Respiration is re-established: at first it is irregular, but gradually becomes more and more steady. The cyanosis disappears, and the patient usually passes into a profound sleep. This may last several hours, and he then awakes, feeling dull and fatigued, but otherwise normal. At other times, immediately after the attack, there is vomiting or nausea, and sometimes a feeling of excessive hunger. He may become maniacal, usually with homicidal tendencies, or the post-epileptic stage may be manifested by nothing more serious than some imperative movement, such as running or shouting. The convulsive stage may be replaced by purely sensory phenomena, without complete loss of consciousness, or there may be merely a fine tremor, or the patient may simply run or be otherwise violent while wholly unconscious. Often there is loss of memory for a longer or shorter interval after the attack (post-epileptic amnesia). The attacks may occur with very varying frequency. In some cases the interval may be years, in others months, weeks, or days; or several attacks may occur in the same day. The severity of the case is to be determined rather by the frequency of the attacks than by their individual violence. If they recur so frequently that one is not completed before the other commences, and the patient remains unconscious for some time, the condition is termed *status epilepticus*. The prognosis is grave.

Petit Mal. In this condition the loss of consciousness is so transitory and the motor symptoms are so slight that its nature often escapes detection. The patient, if talking, will suddenly stop for a moment; there are a peculiar rigidity of the expression and perhaps slight swaying. These will disappear almost immediately, and the patient will resume the conversation. Sometimes after these attacks there will be a feeling of drowsiness for a short period. Auræ may be present in the form of giddiness or twitching of a limb. The attack may also occasionally be ushered in with a scream or a peculiar gasping expiration. Immediately after the attack automatic movements may be performed. Attacks of *petit mal* often occur during sleep, and the only symptoms then that point to the existence of the disease are a feeling of heaviness in the morning, perhaps a sore and bitten tongue, and nocturnal enuresis.

Focal Epilepsy (Jacksonian epilepsy). This form resembles general epilepsy, with the difference that the motor or the sensory disturbances always commence in the same part of the body, and from this part gradually extend until they become general. Thus the thumb may be first affected, showing a tonic and then a clonic spasm; then the hand, the

arm, the whole of that side, or both sides ; or the disturbance may commence in the foot. The disease almost invariably indicates the existence of a focal lesion in the brain.

General Symptoms in Epilepsy. Epileptics are usually dull, apathetic, having a tendency to excess in eating. An excess of indican is often present in the urine. In many cases there is a distinct mental impairment, or, when the disease occurs early in life, there may be congenital imbecility or idiocy. The temper of epileptics is usually irritable, and they are likely to commit acts of violence. They are peculiarly intolerant to alcohol.

Migraine. (Hemicrania.)

This is a disease characterized by paroxysmal attacks of headache associated with nausea and vomiting, and frequently with disturbances of the special senses. The attacks are usually followed by prolonged sleep. The headache is peculiar, in that it commences slowly as a dull but severe pain that gradually increases in intensity, with occasional exacerbations or throbbing, and is limited to one side of the head. Occasionally, however, it is bilateral, but is then usually unequal. At the same time the patient experiences a sensation of intense nausea that may be followed by vomiting. The special senses are affected in various ways. There may be photophobia, hyperacusia, and occasionally the appearance of a peculiar scotoma, which commences as a bright spot that spreads, the outer edge being of an irregular, jagged character, and finally disappears at the periphery of the field of vision. New lines constantly form at the centre, and follow those first appearing. Sometimes the patient complains of dimness of vision, and this may affect only part of the visual field. Occasionally there is temporary aphasia, particularly if the pain occurs in the left side of the head. In addition, the patients may observe vasomotor symptoms, paræsthesia, or occasionally stiffness or spasms in a limb. The paroxysm usually terminates in sleep, which may be prolonged, and when the patient awakens all symptoms have disappeared. Sometimes there is a severe attack of polyuria. The intelligence is not impaired.

Ménière's Disease. (Aural Vertigo.)

This is characterized by attacks of vertigo associated with nausea. The attack usually begins with tinnitus, then intense vertigo, which may come on so suddenly that the patient falls to the ground or is obliged to lie down and remain in this position until the attack is over.

Hysteria.

Hysteria is a disease due to disturbance of the self-control, producing a curious complex of symptoms that appear to be the result of imitation or of a desire to attract attention or sympathy, associated with certain disturbances of the special senses and of sensation. It usually occurs in young adults, especially in women, although males are frequently

affected. There is often neuropathic heredity, and frequently the stigmata of degeneration are present. The psychical symptoms are a certain tendency to self-consciousness, so that the patient is anxious to describe his or her sufferings to surrounding persons; is in the habit of performing ludicrous or startling acts for the purpose of attracting attention; is emotional, weeping or laughing readily, and is often irritable and suspicious. Among the *sensory symptoms* are areas of tactile anæsthesia or analgesia. These may involve exactly one-half of the body, including the accessible mucous membranes, or they may be symmetrical in distribution on both sides of the median line, and often form geometrical figures. They are not the result apparently of simulation on the part of the patient, because they remain unchanged for a number of days. Tenderness—that is, hyperalgesia—may be present over the ovaries and the spine. The areas of anæsthesia may be transferred from one part of the body to the other, either spontaneously or as a result of suggestion. The latter is most effectual when the transfer is made by means of a magnet or metals. The special senses may have their function exalted, so that the patients have an extraordinary acuteness of smell or hearing, or find it difficult to endure strong lights. Depression of the function of the special senses is perhaps more common, particularly loss of the sense of smell and of taste. Hysterical deafness is exceedingly rare. Hysterical blindness not infrequently occurs, and is characterized by widely dilated pupils which usually react to light, and by normal eye-grounds. The hysterical stigmata associated with the eye are of great importance, partly on account of their peculiarities, partly on account of their persistence. The most frequent is simple contraction of the formed field. This, however, occurs in other conditions, and is therefore not so characteristic as contraction of the formed field with inversion of the color field—that is to say, a red object will be seen further from the central visual point than a blue one. Monocular diplopia, in the absence of structural defect in the eyeball, is pathognomonic of hysteria. In rare cases three images may be perceived.

The *motor symptoms* are paresis, or occasionally complete paralysis. The commonest form of this is *hysterical aphonia*, in which the patients are unable to contract the vocal cords for the purpose of producing sound, but may be perfectly able to cough or perform any other function with them. In these cases speech usually returns suddenly under the influence of a strong emotion or suggestion. The paralysis in other parts of the body occurs in imitation of some form of organic disease. Thus there may be paraplegia, hemiplegia, or monoplegia. Loss of power is rarely complete, and occasionally patients move the limbs when they believe themselves unobserved. The electrical reactions remain normal, although the degree of resistance in the skin may be greatly increased. The reflexes are exaggerated, especially those due to cutaneous irritation, such as the plantar reflex, but ankle-clonus does not occur. The gait may be staggering, imitating cerebellar ataxia or the ataxia due to intoxication; sometimes there are tremors, coarse and irregular, and rarely constant. In some cases of hysteria actual contractures of the muscles occur, indicating the existence of trophic disorders. Spasmodic contractions

sometimes occur in the muscles of the abdomen, giving rise to an apparent or *hysterical abdominal tumor*. Actual trophic changes may also occur in hysterical patients, but these are rare in this country. There may be hemorrhages into or from the skin, particularly from the forehead, palms of the hands, and soles of the feet (*stigmata of the Passion*), or there may be localized areas of gangrene of the skin.

The *attack* (*crise hystérique*) may be divided into the prodromal period and the convulsive. The auræ consist of a variety of sensory disturbances, of which the most common is the sensation of a ball rising in the throat (*globus hystericus*). The patient may also have a sensation of heat or cold, or moisture of the skin, or various painful impressions. Occasionally the tenderness over the ovary is greatly increased (*ovaria*), and the attack may be precipitated by pressure in this region. It is impossible to describe all the movements that occur in the *grande crise*. The convulsion may be tonic or clonic. The patient may assume the most extraordinary positions. Among the most characteristic is opisthotonos, in which the heels and back of the head rest upon the floor or bed, while the body forms an arch; or the patient may assume attitudes that suggest or are characteristic of mirth, sorrow, fear, passion, etc. *Catalepsy*, a condition in which the limbs are plastic and remain in any position in which they are placed, may supervene. Consciousness is rarely entirely lost, although there may be subsequent total amnesia for the period of the attack, and, no matter how violent the movements of the patient, injury to any part never occurs. Gradually the movements become less violent, the patient becomes quiet, and consciousness returns. During the attack the pupils are usually dilated, the reflexes may be increased, and respirations are commonly extremely rapid—in one case that I observed they reached 100 a minute. Occasionally the attack may be cut short by pressure upon one of the hysterogenic zones. After the attack the patient may be perfectly normal. At times there may be persistent, perverse tendencies, such as unwillingness to eat, or, at least, a simulation of fasting.

Neurasthenia.

Neurasthenia is a disease characterized by an exceedingly complex symptomatology. The most common general symptom is a subjective sense of fatigue, both mental and physical. The symptoms may be divided into the general and special groups: the former including those common to all forms of neurasthenia, the latter those associated particularly with subjective and objective functional disturbance of the various organs. The mental symptoms are various. The patients are usually querulous, depressed, and hypochondriacal. They are very irritable, but incapable of prolonged emotional exaltation. They find difficulty in concentrating their attention, particularly upon those subjects with which they have previously been familiar. Memory is impaired, and the intellectual capacities are apparently diminished. It must be remembered, however, that careful testing of the memory or judgment rarely shows that it is seriously affected. An important symptom is the insomnia. This may

be of all varieties, but ordinarily the patient, after sleeping in the early part of the night, will awaken and be unable to sleep again for some hours. The statements by the patients in regard to this symptom are very unreliable. Frequently they complain of unpleasant or frightful dreams. Among the sensory symptoms the most important is headache. This is of a peculiar but almost typical form. The patient complains of a heavy, dull feeling, as if wearing some heavy object, the usual simile being a leaden helmet. Occasionally the pain is localized—sometimes to the occipital region and sometimes to a circumscribed area, the latter usually the result of suggestion. Another symptom that is very common is pain in the back. This is usually felt in the neck or in the lumbar and sacral regions; it is of a dull, persistent character, and may be associated with points of tenderness over the spine. Occasionally there are disturbances of the special senses. The patient may complain of inability to see sharply, or there may be *muscæ volitantes*. At other times he will fail to hear distinctly, or may complain of roaring or tinnitus. Actual diminution of the visual power or of the sense of hearing does not occur. The patients may complain, however, of *paræsthesiæ* in the limbs and of various symptoms usually the result of suggestion. Sensation is otherwise normal. There is usually a general decrease in muscular power. Sometimes this may be preserved for short periods of activity, but fatigue, as a rule, comes on very rapidly. At other times it is impossible for the patient to exert the amount of force that would be normal for his muscular development. Occasionally this weakness is localized to one limb or side of the body. When the patient is directed to hold a limb rigid or to extend the fingers forcibly, a fine tremor of the extremities is visible. This may be persistent or readily exhausted; in addition, fibrillary twitchings of the muscles not infrequently occur. The tendon-reflexes are generally exaggerated. Ankle-clonus, however, excepting the form spoken of as pseudoclonus, is exceedingly rare. Absence of the knee-jerk does not occur in neurasthenia. The cutaneous reflexes are sometimes greatly exaggerated, sometimes decreased. Vasomotor symptoms are very common. The patient flushes easily, and there is often dermographia; he complains of palpitation and occasionally of irregularity of the heart's action. Tachycardia is not uncommon. Often perspiration is produced by slight exertion.

In addition to these symptoms, the neurasthenic may complain of various local disorders of the nervous system; he usually suspects that he has locomotor ataxia, and he will probably have learned the symptoms of this condition sufficiently well to imitate them more or less accurately, or he may believe himself suffering from general paresis or brain tumor, or any other condition with which he may be familiar. From general paresis the diagnosis is sometimes quite difficult unless the Argyll-Robertson pupil, which never occurs in neurasthenia, is present. Another common form is gastro-intestinal neurasthenia. The patient may complain of excessive acidity, and, in fact, vomit from time to time masses of acid material, or there may be difficulty in digestion and hypochlorhydria or anacidity. Constipation is an exceedingly frequent symptom. From time to time the patient may also evacuate large quantities of mucus, and

sometimes there may be persistent mucous diarrhoea. This is one of the most intractable forms of the disease. Finally, the patient may be a sexual neurasthenic and believe himself suffering from organic or functional disease of the genital organs. In this variety the various types of sexual perversion are usually, but I believe incorrectly, included. The degree of neurasthenia is spoken of as mild or severe, according as the symptoms are slight or pronounced.

Traumatic Neurosis (Railway Spine).

This can hardly be described as a clinical entity. Some of the cases are unquestionably hysteria; others partake of the nature of neurasthenia; others are instances of pure simulation. Not infrequently all these factors are united in one patient. The usual course of these cases is as follows: After some severe accident, with or without demonstrable injury, usually of such a nature that a claim for damages can be made, the patient begins to complain of various paræsthesias in the supposedly injured part, usually the back; these gradually increase in severity, may be associated with motor disturbances, such as tremor, spasms or paresis, and occasionally with objective sensory disturbances. At the same time there is insuperable dislike for any form of sustained work, irritability of temper, more or less severe insomnia, and occasionally impairment of the general nutrition. Tremors often of the most severe form are present, usually elicited by voluntary movement or the presence of observers. They may be associated with the simulation of intense suffering. The tendon-reflexes are usually increased, the skin-reflexes sometimes diminished, the sexual power impaired, and the visual fields contracted. The patient may complain of palpitation, and the pulse may be high. He devotes himself to the study of his symptoms, to the exclusion of other forms of mental occupation. The course is variable, but in the great majority of cases more or less improvement occurs as soon as damages have been obtained. Nevertheless, some cases do not improve under these circumstances, and others occur under circumstances that preclude any possibility of recompense. The diagnosis should never be made until the patient has been under skilled observation for a considerable period.

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